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THE ARTHROPATHIC DYSTROPHIES.

An Essay

Historical, Experimental, & Philosophical.

Der ruhigste naturwissenschaftliche Beobachter ist nicht immer ganz sicher, dass seiner Wahrnehmungen sich nicht unmerklich den Anschauungen anpassen, mit denen er an seinen Gegenstand herantritt.

(Kraepelin)

Introduction.

Few subjects interest modern pathologists more than that of muscular atrophy in general: its various causes, its different clinical manifestations make it a subject which has powerfully attracted some of the greatest minds in the profession. It is only, however, within the last fifty years that systematic investigation has been carried on both as to its clinical and pathological aspects.

Perhaps we owe more to Duchenne of Boulogne than to any other workers at the subject for the first clinical classifications; before him muscular atrophy was vaguely included in and considered part of - and indeed a result of - "paralysis." By his communications to l'Académie des Sciences in 1849 & 1859 he drew the attention of the profession to the subject,

and since the latter date progress, if at first slow,has at any rate been steady. By Vulpian and his pupil Prevost the first great step in the pathology of the subject was made 1870. It must be well understood,however, that previous to 1849 muscular atrophy had been recognised as such by such men as Abercrombie, Charles Bell, Graves, Cook, Darwall, of England, and Van Swieten and Dubois on the Continent; indeed in 1858 Roberts quotes cases reported by Parry in 1828 and Mayo in 1836. I have however no intention of giving a history of muscular atrophy, which would hardly be sufficiently germane to my subject. If, however, muscular atrophy in general has attracted so much interest, one small department (the name of which heads this paper) has, except in France, been strangely neglected. In its practical importance it stands second to none; its treatment and its prognosis are most important, and its relations to other muscular atrophies are of the most interesting. The mechanism of its production is still unknown, in spite of earnest and good work on the subject.

The opportunity afforded me by a visit to three of the principal medical schools of Europe, to make some small addition to the mass of general knowledge, I have endeavoured to employ by a study of this subject; and if in the course of my work I have made no notable discoveries, but merely placed the work of others on a basis if possible more firm than previously, I still shall

have the satisfaction of increasing the number of those acquainted with their work.

In choosing the title for this paper I have dropped the word Amyotrophy and substituted for it the word Dystrophy, because, - and this is a point I insist on & which no previous experimenter appears to have considered of importance - the other tissues of the limb, that is the bones, the cutaneous and subcutaneous tissues, the nerves themselves, have all undergone changes in their nutrition.

These dystrophies are found in all kinds of arthropathic processes, such as acute arthritis, tubercular disease, fractures of bones into a joint, chronic arthritis, and also in rheumatism, acute, chronic, and septic (gonorrhoeal); in rheumatoid arthritis and Tabetic arthropathy, (Charcot's disease), though in the last two cases the mechanism may be different, unreduced dislocations, etc.

My best thanks are due to Professors Raymond and Stricker for their generosity in allowing me to work in their laboratories at the Salpétrière and the Institute for Experimental Pathology, University of Vienna, respectively; to their chief assistants, M. Najeotte and Herr Biedl, without whose interest and assistance my work would have been much more arduous. Further, I must thank Prof. Sidney Martin for his permission to finish my work in University College, London.

The Drawings are intended as diagrammatic representations in one plane of what is seen in the microscope to be in several planes. Photography without such assistance could easily be used rather to mislead than to illustrate. They have been tinted uniformly with methyl blue, and no attempt has been made to imitate the colour which a preparation stained with Thionin assumes. Thionin itself used as pigment for drawing is of a blue tint, which bears no resemblance to the beautiful purple it gives protoplasmic tissues.

In the technical part of the Photographic work the assistance of my brother, Mr J.A. Clinch, has been invaluable.

The instruments used throughout have been a Watson's 1 inch and 1/6 inch objectives, and a Reichert 1/12 inch oil-immersion objective. Eye pieces and stand by Watson.

Part I. Historical.

CHAPTER I. Historical resumé.

The occurrence of weakness and wasting after joint disease was recognised by Hippocrates;[1] under diseases of the shoulder he recognises atrophy of the deltoid as a sequel to long-standing conditions. Atrophy of the forearm occurs after dislocations of the fingers if not reduced, and dislocation of the femur, in those not fully grown, and if left unreduced, leads to loss of flesh and muscle. The segment nearest the diseased articulation is always the most affected. He explains it as resulting from want of exercise, a cause which naturally acts more on the parts nearest the disease.

Galen,[2] according to Deroche, misunderstood the condition. Personally I have not succeeded in finding any reference to it in Galen's works. Petit[3] describes wasting of the limb as resulting from fractured bones. Hunter,[4] (1835) recognises that the results of sprains last for years- even 50 years or over. "We know that at last sprains are seldom cured." He apparently presumes that this slight tendency to healing is due to injury to cartilage, for he says,"Where there is a perfect cure obtained, I suspect the ligaments only have been injured, and not the cartilage, which is extremely slow to restoration." To the muscular atrophy itself he gives more space than a modern text-book; he says "so that the muscles appear to sympathise with those parts of little ac-

tion, and becomes wasted and weakened in consequence; I think this arises from sympathy or a consciousness of the parts being unable to answer to the action of the muscles, and it comes nearest to human reason of anything in the body." Further on he says, "When the muscles waste in consequence of disease in the joint the surgeon will often say it is from want of action, but if he will only observe the muscles of the other leg, nearly of their full size, though they have had no moremotion than the muscles of the diseased leg-"

Nelaton[5] (1835), about the same time, in relation to coxalgia, attributes the wasting to immobility, and also points out there is also an atrophy of the whole limb, including the bones. It may be that Nelaton, dealing with cases of coxalgia, was dealing with undeveloped persons, and that the atrophy (sic) was merely arrested development, but in any case it points to a profound nutritional disturbance.

Godin,[6] (1835), points out atrophy of muscles round the shoulder following chronic disease thereof.

Coulston,[7] (1837) says in reference to flattening of the buttock in Hip-joint disease that "it is evidently the condition of the glutei which causes this flattening of the hip in the erect position, the total inactivity of these muscles causing them to waste. The muscles of the leg become also affected and its power is greatly diminished." (N.B. He gives no explanation of this.)

He quotes Mr Tyrrel (St. Thomas' Hospital Reports Vol. I) 'as soon as the capsular ligament becomes affected we have sympathetic affection of the knee, and sometimes sympathetic affection of the muscles, at a great distance from the part which is the immediate seat of disease'; and then explains this by saying that "from the intimate connection of the long head of the rectus femoris with the outer head of the acetabulum and with the capsular ligament, this muscle may take on the inflammatory action, and the pain in this way be conveyed down the limb to the thigh." Bonnet,[9] (1838) attributes the wasting to immobility. Malgaigne,[10] (1847) says that in unreduced dislocations many - especially the inactive muscles - atrophy (note not only the inactive); they undergo fatty degeneration." Here he was evidently misled by the colour of the muscles. Roux,[11] (1845), in describing a case of atrophy of the deltoid following hydarthrosis of the shoulder, states that he believes this has the same origin as the atrophy of the abdominal muscles seen after long-continued Ascites, that is to say the muscle has atrophied from pressure. Cruveilhier,[12] (1855) says "the great law which presides over all amyotrophies is immobility, or the absence, more or less complete, of muscular contractions, whatever the cause may be; absolute muscular atrophy supposes a state of absolute immobility, for if a little of a muscle contract, its nutrition is maintained at least as muscular fibre." He says later, "It is probable that nervous action ex-

? hydrarth-

ercises on the muscles relatively to their atrophy a double influence,- 1st, a direct action on their nutrition; 2nd, an action indirect by immobility to which the muscle is condemned in consequence of the absence of nervous influx; but it is well demonstrated that immobility pure and simple, independently of all nervous lesion, suffices itself to determine muscular atrophy."
[13] Adams also notes muscular atrophy as a part of rheumatoid arthritis. [14] Charcot, [15] Palaisance, (a pupil of Charcot) and others touch upon the subject at this period in various relationships. [16] Gubler, (1860) classes the atrophy of rheumatic fever with that seen in typhoid and diphtheria. [17] Bauer (1859) considers the atrophy in Hip Disease a more rapid one than mere inaction would produce, and refers it to reflected nervous action.
[18] Brown-Sequard, (1860) considers that they are of reflex origin of vaso-motor character. [19] Vulpian, (1866) discusses it for the first time, and agrees that it is of reflex character but not vaso-motor. The same year [20] Vergely describes its occurrence in chronic rheumatism.
[21] Liègrois (1869) discusses the pathogeny of articular amyotrophies. [22] M. Ollivier (1869) says that the muscular system feels the effect of (épreuve le contre-coup) ~~of~~ nearly all the affections which can affect the joints.
[23] In 1870 Mr Paget (Sir J.) discusses the subject, and accepts the idea of a reflected nervous influence. In [24] 1872 M. Le Fort read a communication to the Society of Surgery on the subject, and particularly in relation to

hydrarthrosis of the knee. He however does not discuss the pathogeny of the subject. Collette[25] in the same year discusses periarticular atrophies in Rheumatoid Arthritis. He draws particular attention to the thickening and oedema of skin, which, with the amyotrophy, he attributes to reflex influence. Beni Barde[26] (1872) and Sabourin[27] (1873) reproduce the idea of Lasègue that it is due to a rheumatismal inflammation of periarticular fibrous structures. Weir-Mitchell[28] (1874) accepts the theory of reflex origin. In the preface of the French translation Vulpian[29] says, "A group of anatomical elements of the cord are struck with inactivity following an injury which occurred to a part of the body that appears to have no direct relation with these elements. Hence a paralysis of the muscles animated by the nerves whose action necessitates the integrity of these elements." In 1875 Vulpian makes a further reference to them, and again decides in favor of their reflex origin. He says "that it must be included among the so-called reflex atrophies, that is to say among those which are the result of a modification produced in some one or other portion of the grey matter of the cerebrospinal centre by irritation of the peripheral extremities of certain sensory nerves, thereby enfeebling the anatomical elements of this part of the grey matter of the cord." Desnos & Barié[30] (1875) report a case of atrophy resulting from a traumatism. In 1876 M. Le Fort recommends their treatment by galvanism as opposed to Faradism and Massage. Onimus[32] discusses the subject in

the same year. 1877 Mr Paget (Sir J.) makes the following remarks:- "It is, I repeat, no mere wasting from disuse; it is far more rapid than that x x x x and is, I think, very rarely, if ever, so well repaired as is the wasting from disuse. x x x x. I wish I could explain it better than by calling it reflex atrophy. It seems dependent on disordered nervous influence." Further on he says its presence is not pathognomonic of organic joint disease. Vulpian discusses the subject again this year, but there is no change in his previous views.

The great work of the year on this subject is, however, without doubt that of Valtat. Up to this moment all work done had been clinical and conjectural. Valtat, a pupil of Le Fort, and to whom Le Fort gives the entire credit of his work, placed the matter for the first time on an experimental basis, and by a series of carefully carried through experiments largely improved our knowledge. He showed that in animals a pronounced atrophy always follows an arthritis, and by careful microscopic work showed that this is independent of myositis or neuritis or myelitis. He attempted, however, to prove more than his experiments justified, and a critic has made the note "Umph! Quelle logique!" beside some of his arguments in the concluding pages of the copy in the Paris University Library. Darde in the same year wrote a clinical essay on the subject, and pointed out that no relation exists between the severity of the arthritis and the severity of the resulting atrophy. Still

in the same year Berquiem[38] treats of atrophy following coxalgia. In 1878 Urdy and Bocquet have written on the subject, the latter speaking of the suspension of trophic activity of the cells in the anterior horns. Still in 1878 Desplats[39] (de Lille) reviews the subject, and says that neuritis is set up in the nerves supplying the joint; it ascends to the plexus supplying the limb, and then descends the muscular branches. Debove[40] (1880) reports the result of the autopsy of a case: this will be more fully considered later. Picqué[41] (1880), who writes from the surgical point of view, derides all theories but the reflex one. He quotes Virchow as having seen muscles retain their integrity for thirty years of absolute repose. Points out that the neuritis believed in by some has not yet been seen. Christin[42] admits of the reflex origin in rapid cases associated with acute arthritis, but in chronic cases believes the want of use is the chief cause. Batigne[4] arrives at the same conclusions. Descosse[44] refers to anaesthesia and trophic troubles occurring in the neighbourhood of the joint; localises the atrophy to those muscles which are supplied by the same nerves as those supplying the joint. The fatty hypertrophy is usually seen only in rheumatic cases, with which it appears intimately associated; it tends to become more and more marked as the atrophy increases. Vignes[45], still in 1880, refers to its occurrence in rheumatic and gouty conditions, and considers it to be of reflex origin. Dubreuil[46] relates a case of con-

tracture occurring in such a case, and states that it occurs in Brodie's epiphyseal abscesses.[47] Guyon and Féré 1881 relate cases of atrophy of the buttock after slight injuries to the hip.[48] Guichard occupies himself with its nervous pathogeny.[49] Combescure of Montpellier describes various cases of muscular atrophy from causes of peripheral nature, but it is very probable that now many of his cases would be classified as diseases of central nature, especially Syringomyelia. He states that the vastus internus is the most affected. He notes fatty hypertrophy subcutaneously. He draws special attention to the progressive nature of the lesion, with its tendency to invade the whole limb in time, and to the muscular fibrillary contractions.[50] Charcot (1882) quotes Rümpf & Erb and his own experiences, showing that in these cases only a quantitative change occurs in the electrical reactions, and that no reaction of degeneration occurs. He considers there is a stupor of the cell, and recommends strongly the electric spark (static electricity) as treatment.[51] Points out that Hilton had already noticed the occurrence of spasm of muscles in arthritis, and had attributed it to nervous action transmitted through the spinal cord. Points out that the reaction of degeneration is seen in rheumatoid arthritis.[52] Vulpian (1883) reports a case.[53] Barker (1883) in an article in Holmes' & Hulke's System of Surgery merely says that wasting is not observed in the real sense of the word in Hysterical cases. He says nothing about it in relation to arthri-

tis but only in relation to tubercular disease. No hint is given as to its pathogeny.[54] Trelat (1885) reports cases occurring after osteo-myelitis.[55] Duplay and Clado (1885) Progrès Médical, study the histology of atrophies after fractures into joints, and conclude in favor of an irritative process.[56] Moussons (1885) finds nervous lesions occasionally, but not sufficiently often to be able to consider it as cause and effect.[57] Parisot (1886) refers to these atrophies, and concludes for a reflex origin.[58] Desplats (de Lille, 1887) points out that muscular atrophy may follow inflammation of other serous cavities such as the pleura, and records cases.[59] Metje reports the occurrence of atrophies after fractures.[60] Barbillon refers to the disturbances of sensibility seen in such cases.[61] Macnamara (1887) in discussing suppurative synovitis says, "The muscles of the limb become remarkably wasted from myositis." In referring to false ankylosis, "In cases of this kind one has to be cautious about arriving at the conclusion that disease of the bones exists because they appear to be enlarged, a condition not infrequently more apparent than real, and produced by the wasting of the tissues surrounding the joint." This is all that is said on this subject in a treatise limited to diseases of the bones and joints.[62] Strümpell (1888) says that it cannot be referred to the inactivity of the muscle, because from experience we know that inaction of a muscle as such, only in a small degree and very slowly, produces muscular atrophy. But he considers that

reflex action is not sufficient, and that it is really due to direct extension of the inflammation from the joint. He complains that the reflex theory does not explain both the contractures and atrophy.[63] Charcot by a curious coincidence answers this last objection in the same year. I have not the dates of the exact time the two lectures were delivered so that Charcot's may have been intended as an answer to Strümpell's. "The cells of the anterior horn become the seat of an irritative process, which in the first period produces exaggerated reflex excitability of the neuro-muscular system, while in a later period, which corresponds to a period of blunting of the organism, ganglion cell paresis and amyotrophy are chiefly marked. One can understand moreover that in certain cases the blunting, or perhaps the inhibition, predominates from the first, and in these cases it is the paretic and amyotrophic phenomena which from the beginning hold the first place. One can also understand that in certain nerve cells the excitation will be particularly accentuated, while in the others the blunting will be produced early; and thus one explains that at a given moment phenomena amyotrophic and paretico-spasmodic can co-exist in the same member."[64] Spender (1889) in his work on Osteo-Arthritis says "the natural atrophy comes afterwards, for muscles waste when their office is gone."[65] He adopts, it may be mentioned, Ord's view of osteo-arthritis, that "In such cases I have no doubt that the wasting of muscle and skin and the osteo-arthritis

are dystrophies induced by a common central nervous change" (referring to cases in which wasting precedes the arthritis.) But he appears to have advanced no further than Hippocrates did with regard to the pathogeny of muscular atrophies following arthropathies.

In the same year is published Deroche's[66] thesis on the subject, the experimental part of which is merely an excerpt from the work of Raymond published a year later. The work, however, as it stands is of an exceedingly high standard, and well worth study. He brings forward an experiment that by dividing the posterior nerve roots on the one side and then exciting an arthritis in each knee-joint, the atrophy was limited to the side with the intact nerve roots. It must, however, be noted that in the one case given the atrophy is less than is found as a rule under the ordinary circumstances, being here in the bulk of the muscles between 14 & 15%. The dissection was done in a very coarse manner, that is to say large groups of muscles were compared. The Rectus femoris alone comes out with a high percentage loss, viz. 28%. I think it is very probable that division of the posterior root produces disturbances of nutrition in the side affected. The conclusion is of course drawn that the sensory nerves being cut the reflex path is broken, and any dystrophy of reflex character is prevented. Raymond[67] (1890) has conducted a much more extensive series of experiments. In addition to dividing posterior nerve roots in several cases, he has divided the crossed pyr-

amidal tracts on one side, and then obtained a very rapid atrophy. Lesions of the cortex and division of posterior nerve roots also permit rapid atrophy, thus indicating that when the trophic centres were removed both from central and external stimuli, nutrition failed. He also found that irritation of a cutaneous nerve gives rise to exaggerated reflexes, and declares that amputation of a part of a limb gives rise to exaggerated reflexes and atrophy of the remaining part. He describes the following train of symptoms as following on an experimental arthritis:- 1st. Functional impotence lasting a few hours; 2nd. Exaggeration of the knee reflex; 3rd. Exaggeration of the cutaneous reflexes. This is associated with increase of excitability of the cortical centres. 4th Exaggeration of the idio-muscular contractions; this is seen to occur even when the animal is curarised. 5th, Exaggeration of the faradic excitability; 6th , Troubles of sensibility; hyperaesthesia, never anaesthesia or analgesia - this always affects the whole limb. 7th, Muscular atrophy, which invades successively (presumably in lesions of the knee) the Quadriceps, extensor and glutei, the muscles of the leg. It is most marked in the superficial bundles and least in those near the bone. There is increased excitability of the cortex to faradism, and also of the crural nerve to faradism.

Allingham[68] (1889) in a work on internal derangements of the knee-joint does not mention muscular atrophy at all.

Ballet[69] reports a case of severe muscular atrophy in

hysterical coxalgia.[70] Darkschewitsch has reported the histological investigation of a case. This is referred to elsewhere.[71] Brackett (1891) has a most vigorous paper attacking the reflex theory most fiercely, as he does not think it sufficiently explains matters. The degree of atrophy is so various; atrophy of bones even occurs in long-standing cases. It bears no relation to the severity of the injury or its duration. (c.f. this and the previous sentence.) He says we must disregard the cases running a severe course, because in them are all the conditions to be considered. In mild cases so great a difference occurs that there must be a more potent factor in its course besides an irritant nervous influence; cases which have been immobilised will show a decided atrophy - and this in absence of acute symptoms -, and further that similar cases not treated show much less atrophy, but if unmobilised soon show it more markedly, and that the atrophy is proportional to the amount of fixation determined by the apparatus. Clinical evidence does not, he says, bear out the statement of the experimenters that the extensors are chiefly affected. Duplay and Cazin[72] (1891) record a most carefully examined series of experiments in which the arthritis varied in length from four or five days to twelve months. The irritant used was a 10% solution of nitrate of silver. They found marked atrophy at the end of four days. Their histological results are noted in another chapter. Gowers[73] (1892) states that it is the extensor muscles that

chiefly waste, that atrophy occurs in all kinds of joint inflammation and almost invariably. In very rare cases it affects all the muscles of the limb. He considers initial palsy produced by arthritis, apart from the pain, to be rare, and states that contracture is rare and occurs in the antagonistic muscles. He reports a case in which an arthritis in the knee and ankle gave rise to spastic Paraplegia. [74] Lane (1892) on rheumatic diseases refers to the wasting of the muscles of the ball of the thumb, and the interossei as being of diagnostic value between rheumatoid and rheumatic arthritis. He then proceeds, "I am of course omitting any muscular atrophy caused by enforced idleness on the part of the patient, and am assuming that fair joint power has been maintained". He makes no comments on the pathogeny of the muscular atrophy that may occur in these diseases. [75] Charcot (1893) draws attention to amyotrophies occurring from ab-articular lesions. [76] Ferrier accepts the view that these atrophies are of reflex origin, though somewhat doubtful whether a purely dynamic lesion can do so much. Thinks that a lesion of the dendrons of the cells would account for the symptoms. [77] Kornilow (1893) (of whose paper unfortunately I have only been able to see a short abstract) finds the French experiments unsatisfactory and has repeated them, finding that muscular atrophy always occurs on both sides when the posterior nerve root is divided on ~~both~~ one sides. Very probably Kornilow met with some organization of clot or lymph in his medullary canals, with

a result that compression of the anterior nerve roots occurred, but it is hardly fair to criticise before one has seen the entire work. Mansell Moullin[78] in his work m Sprains (1894) mentions wasting of the muscles as a cause of imperfect recovery, but does not give any reference to its pathogeny. Hugnet[79] (1894) gives some cases of traumatic amyotrophy where joints were involved, but prefers to believe that the cause is direct injury to the muscle. Thevenet[80] (1894) reports a case of rheumatic pleurisy (recurrent) followed by atrophy of all the muscles of the shoulder (not the arm) and by flattening of that side of the chest. Hoffa[81] (1894) states that he has repeated the experiment performed by Raymond of dividing the posterior nerve roots and then exciting an arthritis on either side. He arrives at the same results. Plique,[82] the same year, considers that the common sprains of wrist and ankle, however severe, never lead to atrophy. That atrophy is commonest after sprains of knee and severest in those of the rheumatic diathesis, because, however slight the sprain may have been, the constitutional tendency always leads to severe inflammation. Atrophy of deltoid is often really caused by injury to the circumflex nerve, which will yield as a symptom a painful spot at the post border of the deltoid and anaesthesia over the posterior part of the shoulder.(The electrical reaction of the muscle would surely be a better test.) He considers in Hysterical cases absence of amyotrophy is the rule. W.L. Moore[83] (1894) gives a brief

and sketchy account of most of the work done till that date. He presents no points worthy of special comment.
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Marcus Beck (1895) as editor of Erichens Surgery stands remarkably above all other British authors of important works in the attention he gives to the subject. In the last edition considerably more space than in the previous one has been allotted to it. He says, "It is indeed generally believed that the paralysis is induced reflexly as a result of disease of the joint x x ." But still the subject is hardly made sufficiently prominent: for instance, as causes of persistent pain and weakness after sprains he mentions five heads, but the muscular atrophy is at least as important as the last four, and indeed may largely aid in causing them. (They are,- imperfect repair of the torn ligaments, imperfect absorption, strumous, gouty, & rheumatic inflammations, and
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slight displacement.) Bannatyne (1896) in a work on rheumatoid arthritis gives full attention to the subject. He regards the joint condition as a bacterial one, and the recognised amyotrophy as secondary to the joint affection and as being produced reflexly.

CHAPTER II.

Aetiology and Symptomalology.

Opinions differ very greatly with regard to the individual tendency to suffer a muscular atrophy in joint disease. Some authors (Gowers[73] Bazy[86]) say that atrophy always occurs in all kinds of arthritis whilst others (Pheque[82] Le Fort[31], Charcot[63], Brockett[71]) consider that it is rather the exception than the rule. On the whole there can be little doubt that the frequency of its occurrence has been generally underestimated. The universally received opinion that a sprain or a dislocation leaves a permanent weakness of the joint is undoubtedly correct but its explanation that it is due to weakness of the repaired ligaments or to the slowness of repair of the cartilage (Hunter[4]) is probably not so correct as that of Bazy[86] who explains the pain and weakness, as due to the muscular atrophy (he calls the muscles the real ligaments of the joint) weakening the joint and so allowing frequent small strains which keep up slight arthritis and increase the muscular atrophy. Whenever I hear an individual referring to such and such a joint as a weak one, the one he sprained two or three years ago, I ask to be allowed to measure the two limbs and it is rare indeed that I find no difference between them, and this in spite of the fact that muscular atrophy is almost always to a certain extent compensated for by some fatty hypertrophy. Of those however who believe that it is not of constant occurrence, some, (among

them Charcot [63] believe it is due to a neurotic constitution while others (Deroche [66], Raymond [67]) deny this but offer no alternative explanation. Marcus Beck [84] in Erichsen: 9th. Ed. "It occurs chiefly in persons suffering from spinal exhaustion neurotic women and men addicted to sexual excess." It occurs in all kinds of joint disease, dislocations, (Hippocrates [1] Malgaigne [10]) fractures into joints, (Derplay Clado [55]) Brodie's epiphyseal abscesses (Dubreuil [46]) Tabetic arthropathy, rheumatoid arthritis. Tubercular disease generally, Hydrathrosis ((Pheque [82], Le Fort [24]) Acute and chronic arthritis whether a result of diathesis, metastasis, injury, or experiment.

In a simple case of traumatic arthritis at the moment of injury a sudden impotence of the whole limb is experienced in many cases; this may be transient or last some time, only diminishing as the arthritis itself and gradually giving way to the paralysis associated with the muscular atrophy; cases of this kind are according to Gowers [73] rare, experimentally I have never seen them in the adult animal, though Raymond describes it in detail. If in its marked forms this palsy is rare I venture to believe that minor degrees are not uncommon. Personally a sharp blow on an exposed cartilaginous surface, as striking the the elbow or knee when in certain particular positions as against projecting parts of furniture, is immediately followed by a sensation of helplessness, quite independent of the sensation of pain. This passes off in a few moments. Malgaigne [10] describes and explains this as follows. "At the same moment that he feels the first pain, the patient is struck with the impossibility of moving the limb. This

occurs as follows, because the least important movement demands that all the muscles of the limb are set in motion to control the bony levers and because each movement produces lively pains in the displaced muscles." I believe however that the real explanation is that the shock produces such a condition of inertia as Charcot puts it, of the cells in the anterior horns that they are unable to respond to the impulses received from the higher centres. The inflammation of the joint proceeds and the atrophy commences to appear insidiously, progressively, with no febrile or general reaction; according to almost universal opinion the extensors are affect ed first and most seriously. There is no relation between the severity of the lesion and the degree of atrophy. (Deroche [66], Parisot [57], Dard [36], Charcot [63]). The motor paralysis advances proportionally with the atrophy Percussion of muscles reveals higher degree of idiomuscular contraction; the tendon reflexes are generally exaggerated. Generally there is no disturbance of sensibility, but occasionally anaesthesia or hyperaesthesia may be found either in patches or extending over the entire limb, hyperalgesia and neuralgia with painful spots are met with. Hypertrophy of the subcutaneous tissue and skin in some cases hypertrophy (and in others atrophy) of the sweat and hair systems also occur. There is generally Hypothermia of the limb. Contractures are common and may affect the paralised muscles,

their opponents or both. Fibrillary contractions are frequently seen. Thickening of muscular aponeuroses. The subcutaneous fatty hypertrophy is according to Descosse[44] closely associated with rheumatic cases, whereas Clado and Duplay[55] describe it in cases of fractures opening into the joint. Nelaton[5] drew attention to the fact that atrophy of the bones was associated with that of the muscles, a point which appears to have been overlooked by all recent experimenters and workers.

The electrical reactions are of more than passing interest; The Faradic excitability is diminished but the there is no increase in the answer to Galvanic stimulation whose polar reaction likewise remains perfectly normal. Thus we have no reaction of degeneration and this in Charcot's[50] opinion indicates that there is no organic lesion of the nerve cell, and that when it does occur in these cases organic changes are complicating those ordinarily present. In cases of chronic rheumatism and rheumatoid arthritis reaction of degeneration is found but possibly here the pathology is different.

Charcot[50] and some others describe the electric discharge (static electricity) as producing a more powerful contraction than when the muscles are normal.

CHAPTER III.

Pathological Anatomy.

The organs to which special attention has been drawn in this condition are the muscles, the nerves, the spinal cord and the blood vessels.

All observers are agreed that the muscles undergo a process of simple atrophy though the definitions of this process are almost as various as the numbers of observers.

Vallat[35] describes fibres small in number mingled among fibres of apparently normal structure. These fibres are very much atrophied and show granules which are not fatty but are dissolved by acetic acid. These granules are also found from time to time in fibres otherwise normal. In one case he found nuclear proliferation. Moussons[56] next describes loss of clearness of the "Champs de Cohnheim", whilst the longitudinal striation is almost invisible; transverse striation is normal. Duplay and Clado[55] in a clinical case describe fibres with capillaries in their midst, these capillaries having thickened walls. Independent intra-fasimilar fat cells were found. There was irregular moniliform aspect of the fibre, striation was lost at points; they were never normal and some showed a vitreous appearance The sarcolemma is separated occasionally, sometimes it is empty or filled with[25]

is empty or filled with nuclei or fine fatty granules.
Debove[40] compares the changes with those seen in muscular atrophy in cases of hemi-or paraplegia where no disease of the cells in the anterior horn in the spinal cord exists. These having been described by Babinski[88] as simple atrophy of fibre but with increase of nuclei.
Raymond[84] in 1889 considers that in simple atrophy there is loss of the number of fibrils but in 1890[67] believes the atrophy of the fibres is due to the loss of the interfibrillar substance as the longitudinal fibrillation is very indistinct and the transverse sections do not show the typical Champ de Cohnheim, they have lost $\frac{1}{4}$ - $\frac{1}{3}$ their volume. He finds there is no proliferation of nuclei inside or outside of the muscle fibres. The transverse striation is normal. Deroche[66] finds diminution in number of fibrils; increase in interfascicular tissue; diminished appearance of both forms of striation Prohferation of nuclei of sarcolemma. Duplay and Cazin[72] find no sign of irritation. Darkschewitsch[70] describes the appearance he has found in a clinical case. In a certain proportion of fibres mere diminution of the ~~fibres~~ size has occurred, while in a very few more serious changes are seen. The transverse striation is indistinct, the longitudinal is too distinct, with Alum Haematoxylin they stain greenish grey instead of violet blue; they are not granular. Other fibres show a sinuous instead of a straight form..

To sum up we find that the muscles show simple atrophy both with and without granular degeneration and signs of irritation such as increase of interstitial tissue, multiplication of nuclei etc. The only exception to this generalization is the case reported by MM. Duplay and Clado[55] where the lesions are more marked, and where in all probability the condition was somewhat complicated.

In the nerves Moussons[56] finds changes of a degenerative nature which he attributes to the same cause as that of the muscular atrophy. Pitres and Vaillard[90] found neuritis in nerves to muscles but not to the joints. Leloir[91] found neuritis in rheumatic cases. Raymond[67] describes striation of the cylinder axes of the intramuscular bundles by Jakninovitsches method, but he thinks this may be merely a dynamic change. Other authors report the nerves to be perfectly normal.

In the nerves the terminal portions have been frequently found degenerated or inflamed in various degrees The lesions described by Moussons[56] Pitres et Vaillard[90] and Raymond[67] may probably be of the same nature and origin as those of the muscles.

In all uncomplicated cases the spinal cord has been found perfectly normal. The vessels likewise have always been described as normal, barring the case of MM. Duplay and Clado[55] where the capillaries within the muscular fibres are said to have thickened walls.

8

PART II.

Experimental.

After the amount of research already devoted to this subject it would seem at first sight that all the questions to be decided by pure Histology had been answered, but since the last important work (Duplay et Cazin [72]) a method of investigation of the nervous centres owing its origin to Nissl has gradually come more and more into vogue. This method it is now well known reveals lesions which Carmine and Haematoxylin, the reagents in favourite use six years ago, were powerless to show. The question therefore not unnaturally arose in consideration of this matter whether the use of this means of research did not justify a further series of experiments, and the answer was that it did emphatically do so. It was decided to use dogs in these experiments partly on account of expense but also partly because all previous experimenters with the sole exception of Valtat [35] who experimented with ~~both~~ dogs, ~~and~~ rabbits, and guinea-pigs had used dogs, and would therefore afford a sure basis of comparison. Dogs were taken at various ages, from blind puppy-hood to advanced maturity.

Unfortunately a disease of epidemic nature sprang up in the kennel of the laboratory and invalidated to

some extent much of the work; this was as far as was possible neutralised by work at a later period when the epidemic was subsiding. The nature of this epidemic I hope to make the subject of a future work. Briefly it may be said to consist of catarrhal manifestations of the respiratory mucous membranes, the discharges being crowded with abundant bacteria of various kinds, pulmonary inflammations, catarrhs of the alimentary tract and what is of capital importance for this research cellular lesions in the nervous system. It bears a close resemblance to influenza to which disease however bacteriologists say dogs are unsusceptible. (Gunther [92])

The cases divide themselves into two main classes those with septic arthritis and those with aseptic arthritis. In addition two divisions of post: nerve roots one of them followed by arthritis and the other not, have been done.

TECHNIQUE.

The intra-articular injections were done with antiseptic precautions and under heavy morphine narcosis.

To this narcosis is due I believe the absence of the paralytic phenomena observed by Raymond [67] (Whose irritations were also much more severe than mine). The animals experienced no pain at the time of operation and at the end of the first forty-eight hours appeared to have little consciousness of their injury.

They were killed instantaneously by destruction of the medulla, the cord was removed, divided into the necessary lengths, and placed in alcohol 50%. At the end of about eight hours they were further subdivided each vertebral segment being divided into three parts. At the end of the first twenty four hours they were put into 80°/o alcohol and at the end of the second into 96°/o. This was changed every second day till the tenth day when they were put into absolute alcohol and when hard enough passed through the process of celloidin embedding. This last process was not followed in all cases the specimens being cut in absolute alcohol as Nissl recommends, but this process is much more exacting in the degree of hardening and especially in the keenness of the knife used; moreover preparations obtained by its use appeared to show little or no superiority over those embedded in celloidin. After cutting the sections are washed in water and stained in the following (Nissl's [93] formula:-

Methylene Blue B.X. patent	3.75.part
Green Venetian Soap	1.75. "
Aq. Dest.	1000 "

The special Methyline blue indicated is absolutely necessary no others giving anything like the same result and being indeed for this special process almost worthless. [94] Or in a saturated solution of Thionine (Lenhossek) The staining in Methyl Blue is quicker if carried on

at a somewhat raised temperature 60° C. but this is by no means necessary as a more lengthened time has the same effect. The Thionine stains without heat in about ten minutes. It has a very special value: Specimens preserved for any length of time over two months whether in alcohol or in formatin refuse to stain well with Methylene Blue whereas Thionin stains them well- experto crede - after eight months preservation. The sections are then decolorised in 10 o/o solution of Aniline Oil in absolute alcohol, rinsed in absolute Alchohol, Xylol, & Balsam. Nissl's tedious method with resin offers no advantages over the above. Nissl's fuchsin method, as follows, has also been employed. The sections are stained with or without heat in a saturated solution of fuchsin or carbol-fuchsin. Decolorisation in Clove Oil , removal of the clove oil by Xylol, Balsam. This gives very beautiful results, fully comparable with the others, but its great objection is that unless the clove oil is entirely removed by the Xylol, a difficult and extravagant process, the preparation soon fades and becomes worthless. Ladovsky brought forward a modification of the Nissl methods, which he claims produces results of equal beauty with the originals. After staining as previously, he washes in 1 o/o acetic acid till differentiation between the white and grey matter is clear; then decolorises to the exact degree required with absolute alcohol. I cannot find any improvement in this method

93

95

with fuchsin; the decolorisation, slow by the original method, becomes intolerably lengthy, and with the methyl blue the absolute alcohol frequently requires heating - a dangerous and inconvenient procedure, which moreover decolorises the processes of the cells more rapidly than the cell body, so that when decolorisation is complete the processes become invisible.

Weigert's method for the detection of Karyo-kinesis[93,95], a process which Nissl says is always to be seen among the neuroglia cells when the nerve cells are irritated. The preparations are first stained with Weigert's Haematoxylin Solution 1 o/o, then washed and placed in Rademacher's Tincture of the acetate of iron for half an hour, then washed and decolorised in 1 o/o Hydrochloric Acid, in Alcohol 96 o/o, washed, dehydrated, cleared and mounted. This method also gives very fair representation of the nerve cells. I never succeeded in finding any evidence of Karyokinetic changes, but it was not used in all cases. The muscles were hardened in Muller and then embedded in Paraffin, cut and stained with Haematoxylin and Erythrosin, this latter pigment being better from the photographic point of view than Eosin.

The bones were fixed in Muller, hardened in spirit, decalcified in 4 o/o solution of nitric acid (pure), in 96 o/o alcohol, washed in water, hardened again in spirit, embedded in celloidin, cut and stained as above.

Without exception the bones on the diseased side decalcified quicker than those on the normal side.

4 Days.

1. (M) Young dog. Injection into right knee-joint of 1 c.c. of 5 o/o Ag NO_3.

On the fourth day the animal was killed. The limb was swollen and oedematous. Some of the injection had passed into the interstitial tissue behind the joint, and had produced a circumscribed necrosis. The articulation contained excess of watery synovial fluid, the Cartilage and synovial membranes were reddened and injected. The cartilage had not lost its gloss, but the reddening appeared to be due to engorgement of the bone beneath it. Cultures of the joint contents in gelatine and in Bouillon gave negative results.

The majority of the muscles showed much oedema, so that the weights on the diseased side show an increase and not a decrease.

left side		right side			
4·150	Sartorius	3·6	grammes	12·5 o/o	decrease.
7· 05	Rectus	6·5	"	8 o/o	"
30· 83	Biceps	39·81	"	22·5 o/o	increase
48· 7	Quadriceps	72·70	"	33 o/o	"
9· 78	Adductor grac.	10·00	"	2¼ o/o	"
10· 10	Semitendinosus	12·70	"	20 o/o	"
10· 50	Semimembranosus	13·50	"	22 o/o	"

The cervical and Lumbar regions of the cord with their ganglia were examined. Numerous cells on both sides show disintegration of the chromophile granules and hernia of the nucleus; this is more marked in the small

cells in the neighbourhood of the central canal.

It is a phenomenon of so constant an occurrence in all the cords I have examined and in all their regions that no further reference will be made to it in future cases. I assume that it is a post mortem change. In addition to this are found cells with a commencing degeneration of the axis cylinder, along one border of which is extending a fine series of granules. (Fig. 1 & 1a.)

Fig. 1

Fig. 1a

×600

Fortunately in one specimen at least the nature of

Fig. 2

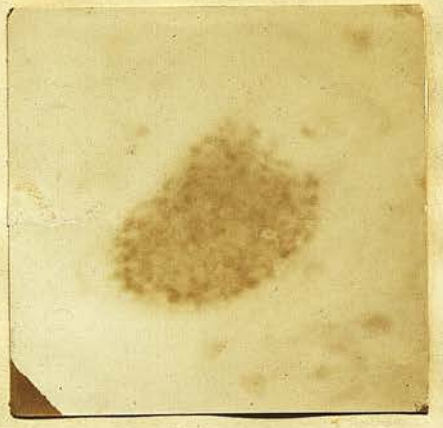
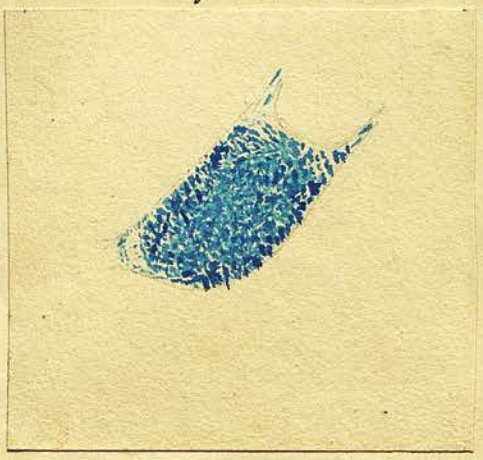
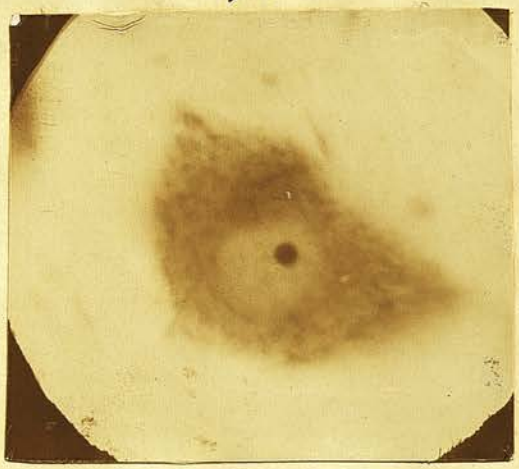


Fig. 2a



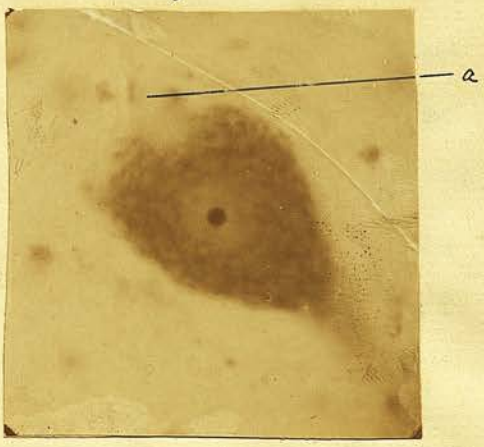
×600

Fig 3



Normal cell. ×600

Fig 4



a. axis cylinder showing superficial granules

the process is unimpeachable, and in this interpretation of the phenomenon I have been supported by experienced microscopists,- notably Dr. Biedl.

The matter is, however, by no means decided. In many cells (Fig. 2) retraction of the kinetoplasm (the name given by Marinesco[96] to the part of the protoplasm which takes on the colouring agent) occurs, and a clear space occurs between it and the cell membrane in which are embedded fine round granules staining deeply. These granules are occasionally found in the clear area from which the axis cylinder arises; but, when they do so, accurate focussing reveals that they are really quite on the surface of the cell and not embedded in its substance. It is to an appearance of this sort that this so-called degeneration of axis cylinder bears a close resemblance, but there is really no identity, the granules in the axis cylinder being continued straight into the granules of the kinetoplasm.

But there is another alternative, i.e. that a protoplasmic process is conjoined for some considerable distance with the axis cylinder, a phenomenon demonstrated by Ramon y Cajal[97] with Golgi's method to be far from rare. It is not likely to be this, because the combination in this case is far longer than is, according to my experience, the rule.

6 Days.

2 (N) Injection of 1 c.c. of a 1 o/o solution Silver Nitrate into the right knee-joint, with antiseptic precautions.

Six days afterwards, when the animal was killed, there was still considerable oedema about the joint and affecting some of the muscles, more particularly the Biceps, the semi-membranosus, the Vasti, and the gastromeneii. Unfortunately merely the difference in weights of the muscles of the two sides was here noted.

				grms
	glutens max.	left is heavier than	right by	•8
	" med.	" "	"	•9
	" min.	" "	"	•5
	?	" "	"	•35
oedema.	Biceps	" lighter	"	2•00
	Sartorius	" heavier	"	•5
	Rectus femoris	" "	"	•6
	Tens.vag.fem.	" "	"	•6
oedema.	Semi-tendinosus	" "	"	0•0
oedema.	Semimembranosus	" lighter	"	•2
	Adductor grac.	" heavier	"	1•4
	Adductor mag.	" "	"	1•5
oedema.	Triceps crural	" lighter	"	14•1
	Adductor long.	" heavier	"	•5
oedema.	Gastroenenius	" lighter	"	1•75

The oedema was characterised by marked enlargement, pallor, non-retractability, and increased weight. The cervical and lumbar regions of the spinal cord and their ganglia were carefully examined, the appearances presented by the cervical being intended to act as a control over those presented by the Lumbar Region.

The spinal cord shows cells similar to those described in the first case, and also cells with retracted kinetoplasm (See Fig. 2.) No difference could be observed between one side and the other either with regard to

Fig. 5

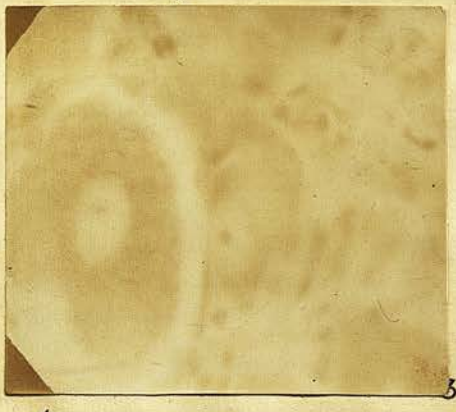
cells showing slight accumulation of kinetoplasm round nucleus: from left side ×600

Fig 6

cells showing centrifugal motion of the kinetoplasm. Left side ×600

the number of cells affected either in the first manner

Fig. 7



from left side. ×600

Fig. 8.

from left side. ×600

Fig. 9

from left side x 600

Fig 10

from left side x 600

or the second, or any other form of degeneration. Both forms of departure from the normal - possibly a normal of too high a standard - are found in the Cervical as well as the Lumbar Region. The first form is more than probably post mortem in nature, as it is chiefly found in that part of the specimen last to be affected by the fixing agent, while the second may possibly represent a cell which either at the time of death or shortly before was actively functioning.

In a similar way departures from the normal are

Fig 11

from right side. x 600

abundantly seen in the cells of the spinal ganglia, but here as a general rule the change is one of much more definite nature. The usual appearance for the cell to take on is shown in Figs. 2 and consists in a change of position of the chromophile granules, which, instead of occupying the cell in a more or less systematic manner, have migrated to the borders, leaving the area between the nucleus and periphery very sparsely occupied. As a general rule, closely applied to the nucleus itself is another ring of chromophile grains. Another change from the usually accepted cell type is when the nucleus is closely surrounded by kinetoplasm, the grains of which become gradually more and more sparse toward the periphery, which may be quite clear. (See Fig. 1).All these changes are certainly within normal limits, and may possibly be connected with differences of function such as is said to be indicated by coarse and fine-grained or light and dark cells. (Nissl.)[98] Further, it may be that they indicate temporary changes in the dynamic potential of the cells. (Flesch.)[99]

8 Days.

3 (E) Small dog of mongrel toy breed, quite young.

An injection of ½ c.c. of Ammonia was made into the right tibio-tarsal joint. Great spasm followed. In a few days disorganization of the joint became evident, but at first remained aseptic; later it became septic, and the animal was at once killed. The use of Ammonia was

suggested by M. Dejerine; it was used in this and two other cases; its action is quite unnecessarily severe, and it leads to complications by infiltrating the muscles in the neighbourhood of the joint. This is, however, of much less serious consequence when, as in this case, it is the ankle joint which is affected. The constitutional effect of such an injection is very slight; indeed all depression, refusal of food, &c. pass off with the morphine. This animal indeed escaped the day before it was killed, and accomplished a running performance, demonstrating that any lesions on the left side of its lumbar cord must be of quite insignificant proportions.

Left	Muscles	Right		
4·485	Glutens maximus	3·32	grammes	26 o/o loss.
12·830	" medius	8·80	"	31 "
1·55	" minimus	1·22	"	22 "
1·60	Sartorius	1·15	"	28 "
4·72	Rectus femoris	3·35	"	29 "
4·72	Tensor vag. fem.	3·3	"	29 "
27·35	Triceps crural	18·75	"	31 "
20· 9	Biceps	14·82	"	29 "
5·42	Adductor longus	5·12	"	6 "
15·07	Adductors -	13·95	"	7½ "
9· 3	Semi-tendinosus	7·455	"	20 "
11·58	Semi-membranosus	8·35	"	28 "
12·17	Gastroenemius	10·22	"	16 "
3·00	Flexor digitorum	2·32	"	22½ "
1·730	Flexor hallucis	·61	"	16½ "
·830	Popliteus	·63	"	24 "
5· 3	Extensor digitorum	4·85	"	8½ "
1· 7	Peronei	1·37	"	19 "
	average loss on right side is			20·7 o/o

The muscles of the right side presented the typical feuille morte colour. An analysis of the weights of the muscles show that the extensors, the glutens medius, the sartorius, the quadriceps extensor and the abductors, the

glutens maximus and the tensor vag. femoris show a higher percentage atrophy than the flexors and adductors.

The spinal cord and its ganglia, both cervical and lumbar, have been examined, but show lesions of a similar nature to those in the two last cases, but in a much more serious and extensive form,- an evidence of toxaemia.

9 Days.

4. (S) A puppy four days old. Injection of ½ c.c. of 5 o/o Silver Nitrate into the left knee joint. On account of the exceedingly small size of the articulation an incision was made through the skin that the position of the injection should be more exactly localised, but at the autopsy it was found that it had by some mischance missed the joint, and a small focus of necrosis was the result. Three days afterwards the entire limb exhibited a marked paralysis, while the opposite one was as lively as is natural in such an animal. In spite of the injection having missed the joint an atrophy of the muscles was very apparent, confirming Raymond's [67] statement that any injury to a limb of a very young animal such as a burn will produce an atrophy and arrest of development;- the difference between the adult and the newborn, in that the muscles of the first exhibit spasm and of the latter a paralysis lasting till death is most interesting and notable. The animal was killed 9 days after the injection.

left (diseased)	muscles	right	
•2	glut. max.	•27 grms	26 o/o loss.
•87	glut. med.	1•07 "	17•75 o/o "
2•69	biceps	2•28 "	15 " gain.
•95	rectus	1•18 "	19½ " "
•41	sartorius	•43 "	5 " loss.
2•83	triceps	3•17 "	11 " "
•820	semi-tendinosus	•853 "	4 " "
2•370	thigh bone	2•620 "	9 " "

Note also the bony atrophy.

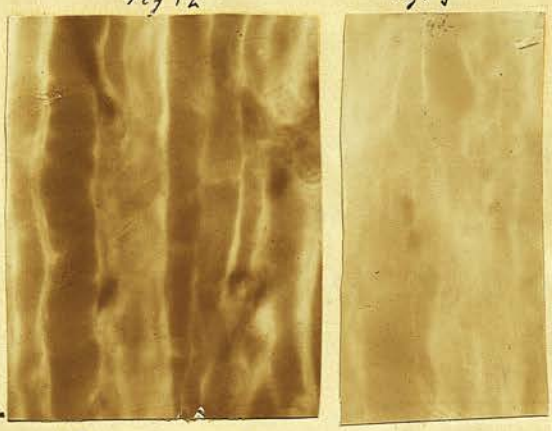
The spinal cord was not examined in this case for two reasons:- 1st, that the articulation was not diseased, and 2nd, that time pressed somewhat on account of the epidemic previously referred to.

12 Days.

5 (T) Similar animal to the last. Treatment exactly the same. This puppy unfortunately died on the 12th day from the epidemic already referred to. After death erosion of the articular surfaces was found; the muscles in the neighbourhood being also necrosed they were not weighed. The spinal cord, on account of the disease from which it died, being of no value for this work has not yet been examined. The muscles show most marked changes in transverse section. Whole groups of fibres can be seen extremely atrophied and with increase of the interstitial tissue in the sections. Some fibres appear enlarged and swollen, and on making comparisons with longitudinal sections we find that they probably correspond with fibres which are at one point atrophied or normal and a short

distance along are extremely swollen, presenting a peculiar varicose appearance. The contents of the sarcolemma are in many cases disintegrated, and show a coarsely granular appearance (x700).

Fig 12 Fig 13



Long. sec. muscle. x 600. shows fibres with irregular calibre. etc

Both the transverse and the longitudinal striation are lost. The nuclei of the sarcolemma are probably increased, but it must be remembered that in the undeveloped animal the nuclei are larger and more conspicuous than in the adult, and that in consequence of the in many parts extreme atrophy of the sarcous tissue a false appearance of increase may be presented. No paryokinesis has ever been observed. The bones also show points of interest, but here the formed tissue is not so readily affected, nor are its slighter changes so readily appreciated as in the case of the muscle. However here the active elements, the Haversian cells, show

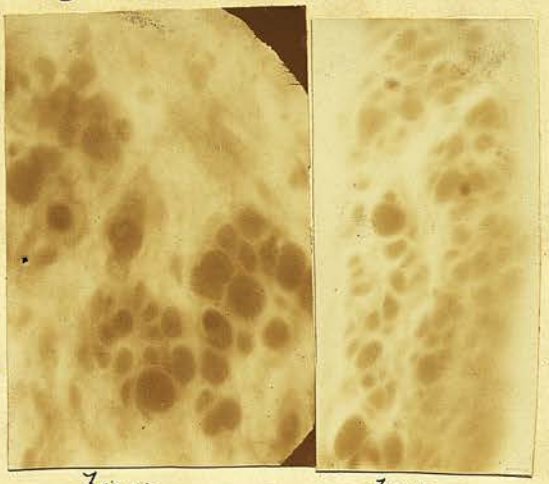


Fig 14 Fig 15

Transverse section x 600

n

Fig 16.

section of bone. n. a cell showing nucleus with changes described.

changes of interest. The commonest change is as follows:- the chromatin network of the nucleus, instead of being evenly distributed over it, is retracted to a strongly coloured mass in the centre, the peripheral part of the nucleus remaining clear and vesicular: at other times one finds the nucleus entirely vesicular and unstained. The cell rarely takes part in these changes, but may be found somewhat swollen. The lacunae in which the cells lie also are apparently larger than normal, and in some cases are empty. The ground substance of the Haversian spaces is more granular, and a more marked tendency is seen to the formation of fat cells. Further under the periosteum and in the Haversian spaces are large coarsely granular protoplasmic masses without nuclei, resembling sometimes a degenerated osteoclast and at others a swollen degenerated and occluded blood-vessel.

Fig 17

normal osteoclast

× 600

Fig 18

protoplasmic mass resembling osteoclast (this is almost certainly due to with a very bright light nuclei can be detected

44. × 600

Fig 19

protoplasmic masses resembling dilated blood vessels.

× 600

12 Days.

6 (Y). Young dog in good condition. Injection of 1 c.c. of a 1 o/o solution of Silver Nitrate into the right knee.

Spasm of the limb producing adduction and violent flexion is exceedingly well marked for the first few days, but afterwards passes off to a considerable degree. Knee jerk is exaggerated on the diseased side. On the 12th day it was difficult for a person who had not seen the animal before to decide which was the affected limb, which indeed was only indicated by a slight stiffness in action as a rule, though from time to time it was carried high. Further, the weight of the body being thrown on to it, it was unequal to the strain. Knee jerk was still exaggerated.

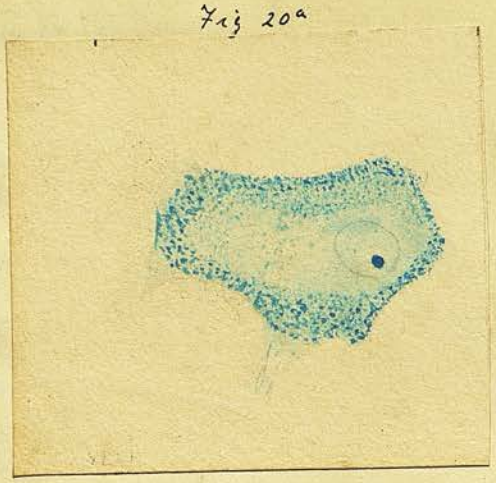
left	muscles	right. diseased.		
2·25	glut. maximus	1·5 grms	33 o/o loss.	
11·40	glut. medius	8·5 "	25½ "	"
17·12	biceps	14·17 "	17½ "	"
1·5	sartorius	1·07 "	29 "	"
3·57	rectus	3·50 "	2 "	"
5·28	tensor. vag. femoris	4·41 "	16½ "	"
25·21	extensor triceps	20·44 "	19 "	"
4·70	gracilis	4·20 "	10½ "	"
24·35	hamstrings	18·60 "	23½ "	"
10·20	gastronemius	9·30 "	9 "	"

an average loss of 18½ o/o.

The examination of the spinal cord and ganglia shows in general much the same appearances as have already been detailed, but in one section a cell was found presenting a typical irritative degeneration, that is to say one cell

Fig 20

Fig 20a



X 600

in 30 sections carefully examined. Here the nucleus has lost its special outline; the area around it has lost its kinetoplasm, which is crowded round the periphery of the cell. (See Fig 20) The muscles and bones present appearances similar to those to be discussed shortly under another case.

12 Days.

(7) (P). This was a dog of about six or seven years of age. Both the right knee and tibio-tarsal joint were injected with 1 c.c. of a 1 o/o solution of Silver Nitrate. The injection was repeated 6 days later, but unfortunately sepsis of the tarsal joint followed on account of the difficulty experienced in thoroughly cleansing the neighbourhood of the joint, the animal being very thin. The animal was killed six days after the second injection.

left muscles		right (diseased)		
1•35	gluteus maximus	•8 g	grms	31 o/o loss.
7•8	gluteus medius	4•7	"	30 " "
13•15	biceps	9•9	"	24 " "
2•300	rectus femoris	1•850	"	20 " "
1•300	sartorius	•95	"	27 " "
2•45	tensor vag.femoris	1•35	"	35 " "

left	muscles	right(diseased)		
20• 58	triceps	17•31	grms	16 o/o loss.
2• 41	gracilis	2•41		0 " "
4• 51	semi-tendinosus	3• 7		13 " "
8• 5	semi-membranosus	6• 1		28 " "

a total loss of about 24 o/o.

The muscles here had a very degenerate yellowish colour. The spinal cord shows similar lesions to Case 3, cells which, while to a great extent preserving their outline, have lost their kinetoplasm. The nucleus is frequently shrivelled or dislocated and the nucleolus stains feebly. These changes are seen throughout the entire cord.

13 Days.

8 (Q). About a year old. The double injection, twice made, was performed as in the last case. The animal died thirteen days after the first injection and seven after the second, from the prevailing epidemic.

left	muscles	right(diseased)		
2• 2	glutens maximus	1•92	grammes	13 o/o loss.
9• 7	" medius	6• 4	"	34 " "
17•87	biceps	13• 4	"	25 " "
1• 5	sartorius	1• 4	"	7 " "
3•62	rectus	2• 8	"	23 " "
4•62	tensor vag. femoris	3•67	"	20½ " "
25•18	triceps	20•17	"	20 " "
4•71	gracilis	4• 2	"	11 " "

an average loss of
~~22~~ 22 o/o

Microscopic examination of the cord showed very pronounced generalised lesions, the result of the disease and not of the experiments. (This I have proved by the examination given to me by other experimenters whose

animals had died from the same disease, but as it is foreign to the subject of this work I give no further details.)

13 Days.

9 (R). A young animal. Injection of 1 c.c. of 5 o/o solution of Silver Nitrate into knee joint. This animal died also from the epidemic.

Left	Muscles	Right (diseased)		
1·770	Glutens maximus	1·600 grms	9½ o/o	loss.
9·500	Glutens medius	7·500 "	21 "	"
18·300	Biceps	18·910 "	3 "	"
2·910	Rectus femoris	2·910 "		"
1·560	Sartorius	1·350	12½ "	"
3·250	Tensor vag. femoris	2·470 "	17 "	"
24·820	Triceps Extensor	23·300 "	6 "	"
4·870	Gracilis	4·620 "	5 "	"
7·370	Semi-tendenosus	6·670 "	9 "	"
12·650	Semi-membranosus	9·880 "	22 "	"
1·000	Adductor longus	·700	30 "	"
15·600	" magnus	11·600 "	25½ "	"

an average loss of 11·75 o/o.

A striking feature in the weights here is the great loss of the adductors,- a quite exceptional occurrence, while the tensors and abductors show a much less degree.

15 Days.

10 (V). A young and well-nourished, healthy animal. Injection into right knee joint of 1 c.c. of a 1 o/o sol. of Silver Nitrate.

The day the animal was killed he showed merely a slight lameness and stiffness of the affected leg. The knee jerk still slightly exaggerated. General health excellent. The articulation showed very slight changes from

the normal; slight increase of fluid, slight injection of the synovial membrane, and a change in colour of the cartilage in the direction of redness, but this is quite imperceptible apart from comparison with the normal knee-joint.

left	muscles	right(diseased)			
3• 4	glut. maximus	2•63	grammes	22½	o/o loss.
17• 5	glut. medius	13•32	"	24	" "
33• 5	biceps	30•00	"	8	" "
2• 4	sartorius	2•00	"	16½	" "
9•	gracilis	7• 5	"	16½	" "
6•16	rectus	4• 8	"	21½	" "
7•25	tensor vag. femoris	6• 2	"	14½	" "
44•70	triceps extensor	34• 7	"	22½	" "
38• 5	hamstrings	•29	"	24½	" "
19•55	gastroenemius	14• 2	"	22½	" "

an average loss of 20 o/o.

The muscles in this case showed very great diminution in calibre of the fibres in general; many other fibres show an extreme atrophy, and some few show enlargement which appears to be a first stage in the atrophy; the lymphatic space around them is dilated. In longitudinal section these fibres may be seen to be of irregular calibre. some fibres show a granular degeneration, but unless this is present neither the transverse nor longitudinal striation appears notably affected. I find no difficulty in noticing the different

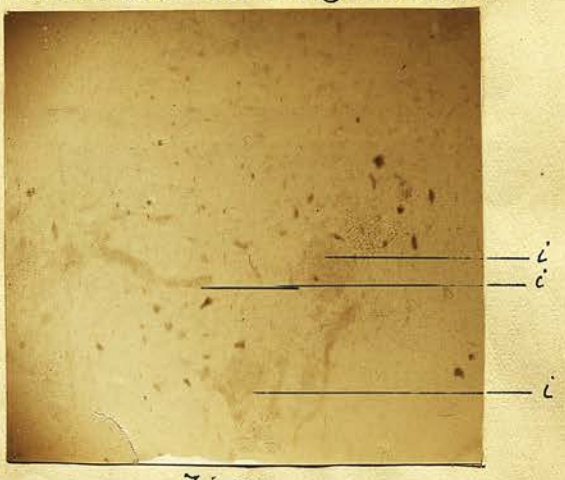
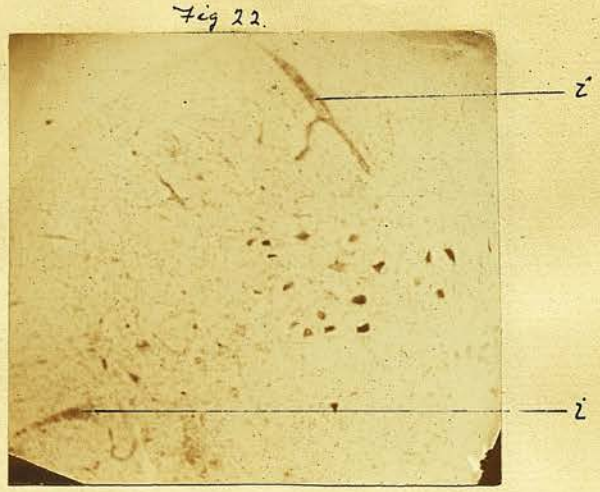


Fig 21
x 33

elements of the field of Cohnheim, and indeed with care they can be successfully photographed. There is some slight interstitial increase. The anterior crural and sciatic nerves showed no sign of neuritis.

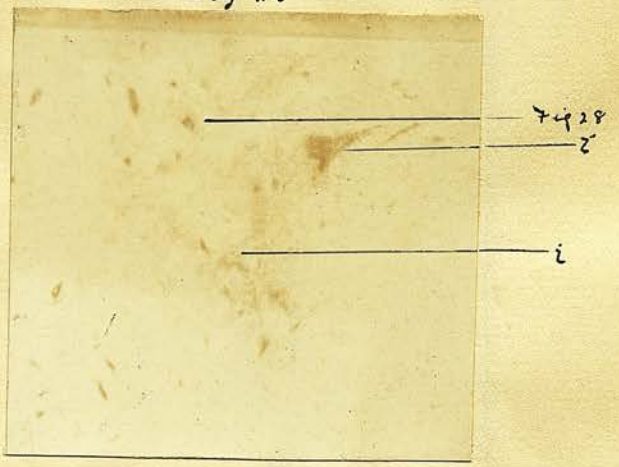
Fig 22.



x 33

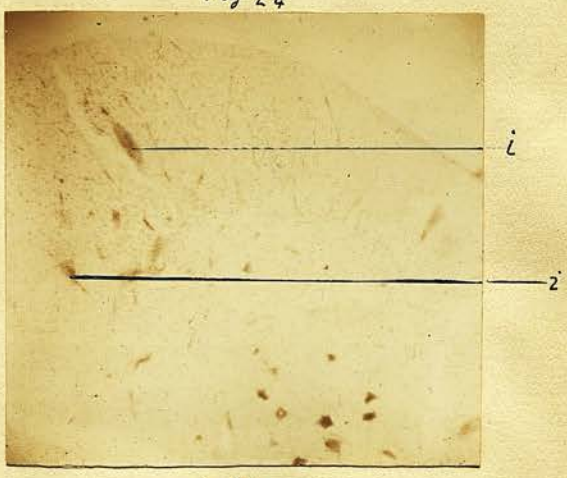
The bones show no recognisable changes. The spinal cord presents here most remarkable lesions on the right side in the neighbourhood of the external group of cells in the anterior horn. They are not seen at all on the left side. Under the low power an area - or in some cases areas - of inflammatory exudation can be seen; they vary in size, and the nerve cells in their midst can plainly be seen to have lost their staining reaction. A higher magnification shows clearly the nature of the inflammatory exudation, the small round cells of leucocytic type.

Fig 23



x 50

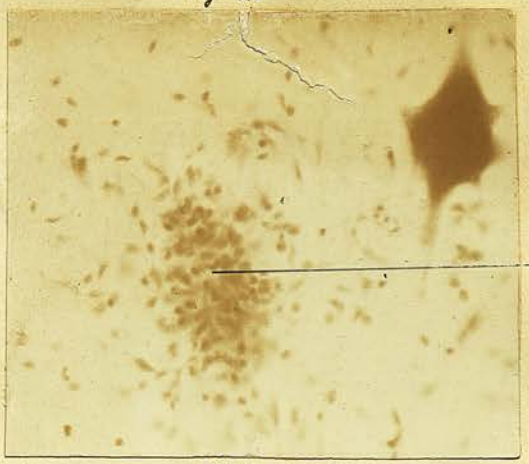
Fig 24



x 33

A higher power still applied to the nerve cells shows

Fig 25



X 300

i. area of exudation.

that the chromophile bodies are broken up into their constituent granules, that the cell body is swollen and its processes frequently lost. The chromophile granules are diffused more or less evenly over the cell, and divide it into areas whose outline can be also traced by fine lines lightly tinted with the staining re-agent. The nucleus also moves to one side or other of the cell, and loses its shape and frequently entirely disappears; the nucleolus may entirely disappear or may take the staining reagent feebly or irregularly; in fact we have a perfect picture of the type of degeneration regarded by Freimann,[100] Nissl,[93] Marinesco,[96] and others as due to a local or primary lesion. The spinal ganglia show no changes apart from those described in previous cases; indeed one cell was found on

Fig. 26

X 600

Shows degeneration of the nucleolus. (The photograph is bad.)

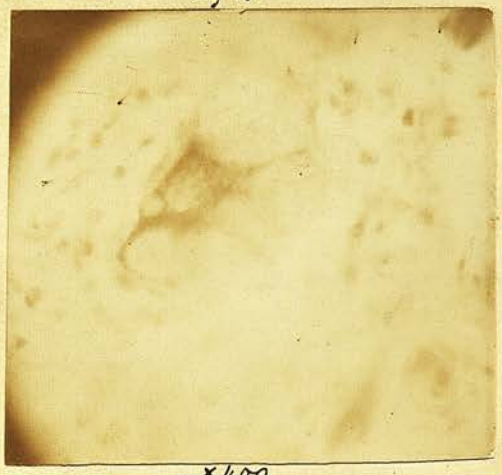
Fig. 27

degenerated cells.

This cell is unimportable & can do well by Photography

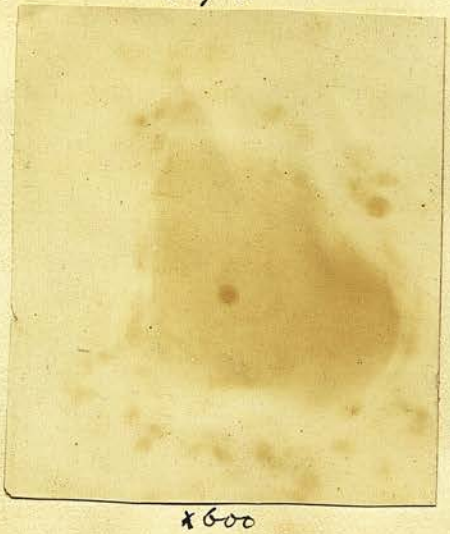
the left side more degenerated than any on the right side.

Fig 28

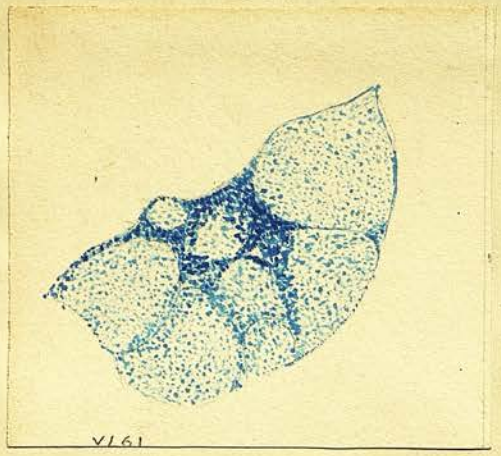


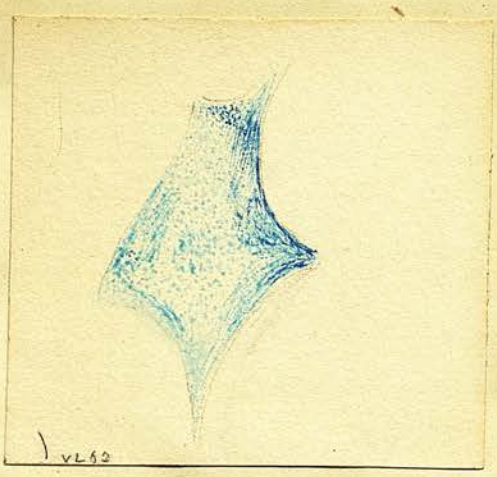
×400
Fig 29

Fig 30



×600





Representations of degenerated cells.

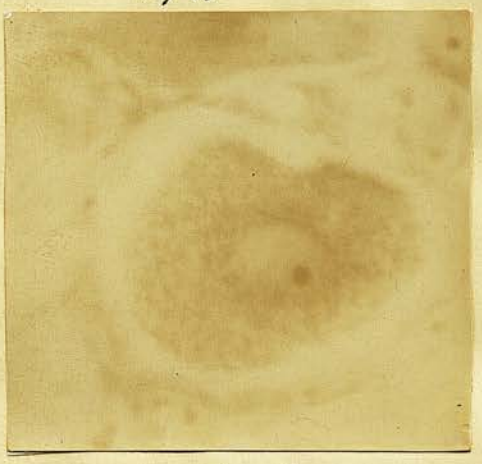
Fig 31

×600
deg. cell from ant. horn

Fig 32

from right ganglia

Fig 33



normal cell. × 600.
Spinal ganglion

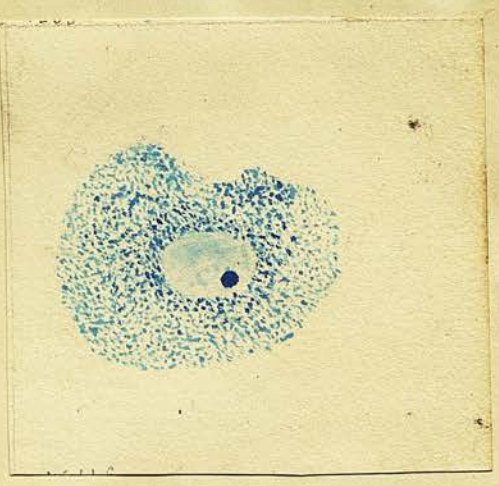
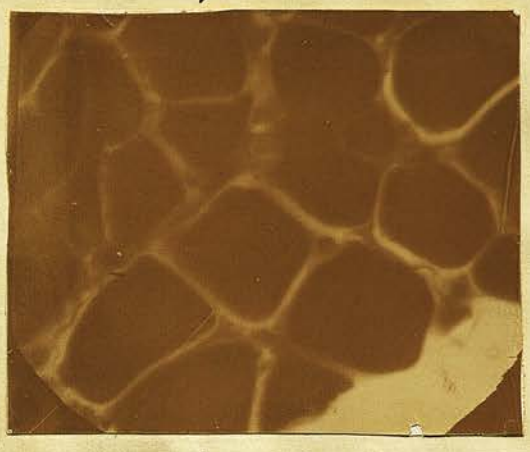
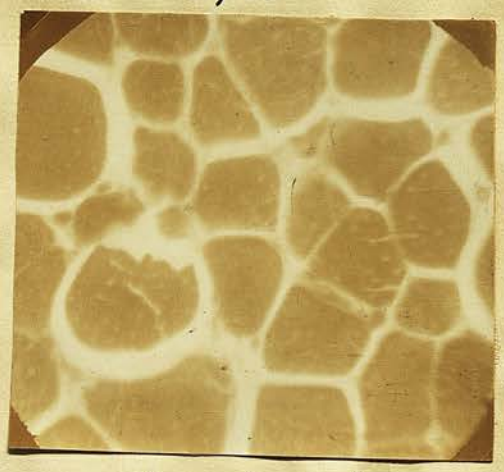


Fig 34



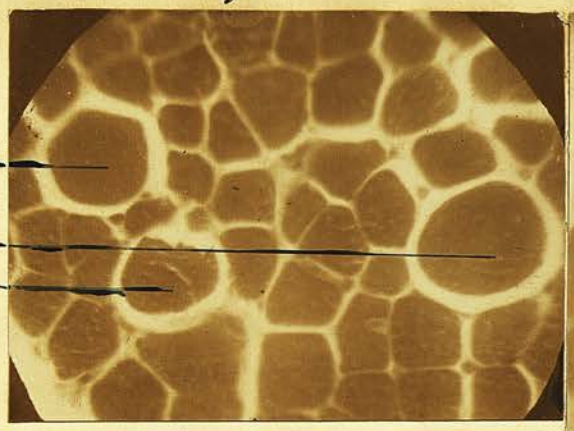
normal muscle × 600

Fig 35



diseased muscle × 60
contrast the average size of the sections
with the normal side.
The "champs de Cohnheim" is well seen.

Fig. 36



abnormal muscle
×400

r. the swollen fibres with dilated lymphspaces.

The general smallness of the fibres can be well seen.

Fig. 37

abnormal muscle
×400

Fig 38.

×600

The longitudinal striation can be seen. The negative shows very clearly the transverse striation but I have not been successful in printing it.

22 Days.

11 (B). Young animal under a year old.

Injection into the right tibio-tarsal joint, 1½ c.c. of a 1 o/o solution of Nitrate of Silver. After the injection the animal carried its leg off the ground, but, if forced to walk, the other leg appeared to lose its strength, and the paraplegic position was adopted. This symptom bears an evident relation to Raymond's[67] paresis, immediately attacking the affected limb. Five days after the injection a peculiar movement of apparently involuntary and spasmodic character was observed, viz. that from time to time the animal stretched the limb out backwards in a jerky manner (the reflex being exaggerated.) A second injection was made 12 days after the first, and 20 days after an injection of 1 c.c. of Ammmonia was made. This at the time produced great spasm of the muscles of the limb. As invariably happened when Ammonia was used, the joint soon became septic, apparently because the track of the needle was never able to close. The animal was killed the twenty-second day.

left	Muscles	Right		
63•23	Triceps	52• 9 grms	16½ o/o	loss.
26•65	Calf muscles	20•55 "	22½ "	"
3• 4	Peronei	2•55 "	16½ "	"
11•75	Anterior leg muscles	8•65 "	26½ "	"

an average loss of 20 o/o.

The spinal cord and muscles here show much the same as has already been noted. The muscles were specially prepared to demonstrate karyokinetic changes, but none were seen.

25 Days.

12 (K). A young black and tan terrier about six months old. Injection of ½ c.c. of a 1 o/o solution of Silver Nitrate into the right tibio-tarsal joint, followed a week later by a second..

A fortnight after the first injection the animal appeared to have suffered but little from the arthritis . There was a certain weakness and rigidity of the limb, and the toes were not properly separated in walking. At the autopsy twenty-five days after the first injection the joint was found seriously disorganised; the cartilage was sloughing, being opaque, greyish, and of the consistence of wet wash leather. No pus or other indication of Sepsis; some serous fluid in the joint. The ligaments were intact and but little affected, and the exposed bone was smooth.

left	muscles	right(diseased)			
6•05	sartorius	5• 3	grammes	12 o/o	loss.
8• 8	rectus femoris	8• 8	"	0 "	"
15• 7	adductor long.	15•35	"	2 "	"
4• 3	adductor brevis	3• 7	"	14 "	"
45• 5	adductor magnus	36• 3	"	20 "	"
33 • 4	hamstrings	28• 2	"	15 "	
77• 6	triceps crural	59•25	"	23 "	
68• 2	biceps	66• 8		2	
17• 3	gastrocnemius	12• 3			
11• 5	flexors of toe	9• 2			
12• 9	ant. muscles of leg	12•			
4• 4	peroneal muscles	3•65			

Here we see again the extensors, which in this case are joined by the gastrocnemius as an extensor of the ankle joint, show a more marked atrophy than the flexors. The pronounced atrophy of the adductor magnus, however,

hardly falls in with the rule that the adductors are as little affected as the flexors. The rectus femoris and the sartorius seem very frequently to except themselves also from the rule, which indeed is by no means of an absolute nature.

Most careful examination of the spinal cord reveals no lesion which could have any relations whatsoever with the experiment. Examination of the ganglia yields a similar negative result.

Fig 89

normal cell. x 600

29 Days.

13. (O). Young terrier about 2 months old. Injection 1 c.c. of 1% solution of Silver Nitrate into left knee. This was followed five days later by an injection into the left tibio-tarsal joint, and nine days after this both joints were again injected. A peri-articular abscess was found a week later, which was washed with 1% Corrosive Sublimate and dressed. At the autopsy, twenty-nine days from the commencement, this was found completely healed; a point in spite of the wonderful healing capacities of young animals appears to negative the idea that it had ever communicated with the joint.

Left	Muscles	Right		
•7	gluteus maximus	1• 0	grammes	30 % loss.
2•75	gluteus medius	5• 5	"	50 " "
6•3	biceps	8• 3	"	24½ " "
1•3	rectus femoris	2•65	"	35 " "
•600	sartorius	•75	"	20 " "
1•55	gracilis	2• 1	"	26 " "
9• 7	triceps	17• 1	"	43 " "
1• 5	semi-tendinosus	3• 2	"	53 " "
3• 6	semi-membranosus	6	"	39 " "
2• 7	gastrocnemius	5•65	"	52 " "

an average loss of 40 %.

The rigidity of the diseased limb was here very marked. The fragility of the bones was also very striking, the tibia first, and subsequently the femur, being broken in the ordinary handling during the course of the dissection. Microscopical examination has yielded no result and throws no light on this matter. The muscles also offer no differences from those already described. The nervous system is similar in this respect. Perhaps it would be well to draw attention to a physiological condition, which if at all exaggerated, may lead to error. It is by no means uncommon in the most normal nervous systems, for the condition may be met with in all parts of it, to find the cells, usually medium-sized ones, surrounded by a number of neuroglia cells, which, stained by a nuclear stain, appear like a

Fig. 40.

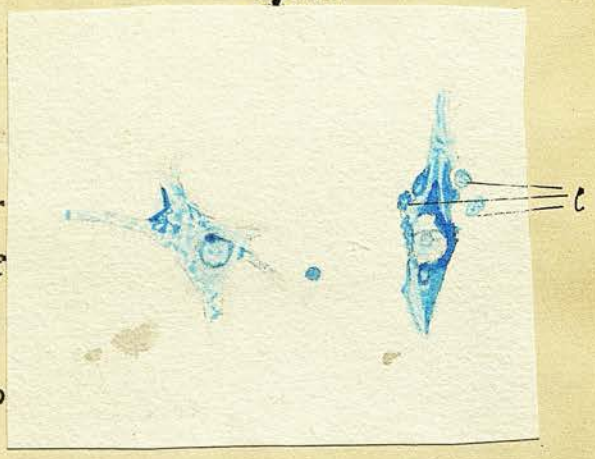


Fig 41

c. The neuroglia cells.
– The nerve cells show post-mortem changes.

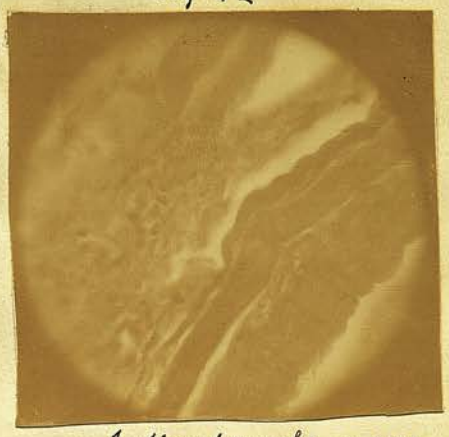
group of leurocytes vigorously attacking the cell. The misinterpretation of this phenomenon has, I believe, already led to error, and it is only so recently as January last that Ramon y Cazal has drawn attention to it. It should be added that these cells may actually make depressions in the nerve element.

14 (A). A mature terrier mongrel. Division of the 2nd, 3rd, 4th, 5th posterior nerve roots in the Lumbar Region (right side.) After recovering from the anaesthetic the animal showed complete paraplegia, paresis of abdominal muscles, incontinence of urine, and faeces. Retained the movements of the tail. This is in accord with the experiments of Sherrington who showed division of a series of posterior nerve roots produced anaesthesia and paralysis. Four days after the animal regained power over the left leg. A month after there was anaesthesia to Faradic electricity of the anterior two-thirds of the two upper segments of the limb, and the knee jerk was still absent. An injection was made into both knees of 1 c.c. of Ammonia. The immediate result was again a paralysis of the left leg, the right leg having remained paralysed all along. Sepsis however soon set in, and the animal was killed a week later.

Unfortunately the sepsis had so destroyed the parts in the neighbourhood of the joint that the muscles were of no value for weighing. Microscopically they show typical acute myositis.

The Spinal cord shows numerous lesions, but they are confined - or almost so - to the right side. The ganglia, on the contrary, show lesions already referred to, but I have not been able to satisfy myself that there is any appreciable difference between

Fig 42



Inflamed muscle. x 220

Fig. 42.a

Fig 42

degenerated cell x 600

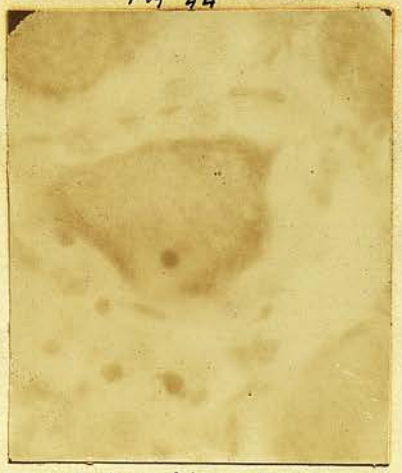
the one side and from the other. The cells in the spinal cord

Fig 43

degen. cell x 600

lose their processes, the nucleus moves to one side of the

Fig 44



cell from left spinal ganglion x 600

cell, becomes distorted and smaller, the nucleolus becomes

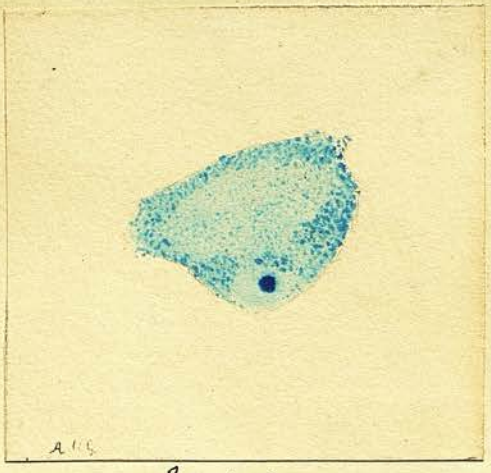


Fig 44a.

degenerated in appearance. The chromophile substance largely disappears, what remains being as a rule clustered round the nucleus. The body of the cell becomes smaller. It is in fact the type of degeneration seen in lesions produced from a distance. (Nissl, Marinesco, Bi[illegible] &c.)

15 (F). Strongly built mongrel terrier. Section of the 3rd, 4th, 5th, and 6th posterior Lumbar nerve roots. This animal resisted the shock of the operation much better than the last one. Haemorrhage, however, set in, but ceased without special treatment. Cutaneous sensibility

could not be ascertained on account of its savage nature. Autopsy 10 weeks later. Certain muscles of the right side appear much atrophied.

Left	Muscles	Right		
5• 5	Sartorius	5• 4	grammes	
18•55	Rectus femoris	7• 5	"	58 % loss.
15•15	Tensor vag. femoris	18• 2	"	13 " gain.
12•65	Adductor longus	9• 2	"	28 " loss.
62• 5	Triceps cruris	43• 3	"	27 " "
42•	Other adductors	34• 4	"	18 " "
27• 1	Hamstrings	30	"	10 " gain.
74• 5	Biceps	76• 5	"	2½" "
26• 5	Gastrocnemius	26• 4	"	
6• 6	Flexors of the Toes	6•45	"	2 " gain.
19	Anterior & external) muscles)	18• 6	"	2 " "

This atrophy is found to be more marked in the pre-axial than in the post-axial muscles. But we find that the tensor vaginae femoris and the hamstrings have atrophied on the other side, and this is due probably to organisation of the clot which resulted from the hemorrhage immediately after the operation.

Fig 45a

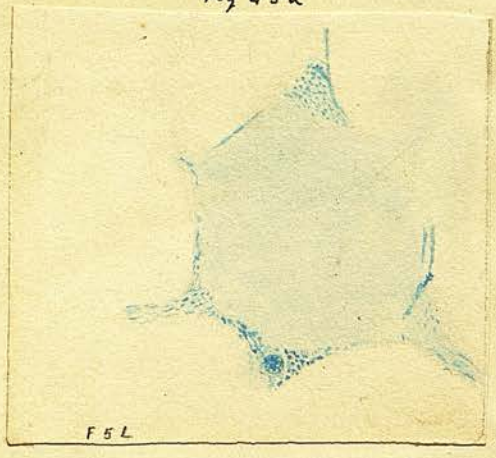


Fig 45

Degen. cell. from ant horn

Examination of the cord has not shown any lesions on the

left side, but merely on the right side; this may possi-

Fig. 46

bly be due to chance in choosing the sections for stain-

Fig 40a

Degenerated cell from Right ganglion

ing. The ganglia show lesions both sides.

Was there no injection of irritant into a joint in this case?

It is impossible to know what value to attach to the results of this experiment owing to ~~wafer~~ incomplete record of the experiment.

	No 11 22 days 1 Joint 3 Injections	No 3 18 days 1 Joint 1 Inject.	No 7 12 days 2 Joints 2 Injects.
Gluteus maximus		26 %	31 %
Gluteus medius		31 "	30 "
Gluteus minimus		22 "	
Biceps		29 "	24 "
Rectus femoris		29 "	20 "
Triceps Extensor	16½ %	31 "	16 "
Sartorius		28 "	27 "
Gracilis			2·41 %
Tensor ~~fascial~~ femoris		29 "	35 %
Hamstrings		24 "	20 "
Adductor longus		6 "	
Other adductors		7½"	
Gastrocnemius	22½ %	16 "	
TOTALS	20 %	20·7 %	24 %

These three cases (all septic) show a remarkable uniformity in their result, although the times differ tremendously and likewise the severity number of articulations affected. It is noteworthy perhaps that No. 7, an old animal with two joints affected and only sepsis of short duration, equals a young dog (No. 3) in the intensity of the atrophy, and surpasses another young dog whose total duration was longer and whose sepsis was about equal duration.

	NO. 1 1 joint 1 injection 4 days Mature	NO. 2. 1 joint 1 injection 6 days Mature	NO. 5 1 joint 1 injection 12 days Mature	NO. 8. 2 joints 2 injections 13 days Old dog
Gluteus Maximus	-	-	33%	13%
Gluteus Medius	-	-	25½%	34%
Biceps	-	-	17½%	25%
Rectus femoris	8%	-	2%	23%
Preceps extremer	-	-	19%	20%
Satorius	12½%	-	29%	7%
Gracilis	-	-	10½%	11%
Senso fascia femoris	-	-	16½%	20½%
Hamstrings	-	-	23½%	-
Gastro-enemii	-	-	9%	-
Adductor-longus	-	-	-	-
Other Adductors	-	-	-	--
TOTAL			18½%	22%

61 a

	NO. 9 1 joint 1 injection (5%) 13 days Young dog	NO. 10. 1 joint 1 injection 15 days Mature	NO. 12 1 joint 2 injections 25 days Young dog	NO. 13 2 joints 2 injections 29 days Puppy.
Gluteus Maximus	9½%	22½%	-	30%
Gluteus Medius	21%	24%	-	50%
Biceps	3%	8%	2%	24½%
Rectus femoris	0%	21½%	0%	35%
Preceps extremer	0%	22½%	23%	43%
Sartorius	6%	16½%	12%	20%
Gracilis	12½%	16½%	-	26%
Senso fascia femoris	5%	14½%	-	-
Hamstrings	17%	24½%	15%	46%
Gastro-enemii	15½%	22½%	25%	52%
Adductor-longus	30.	-	-	-
Other Adductors	25½%	-	16%	-
TOTAL	11¾%	20%	14½%	40%

The above table leaves one able to draw few conclusions. The glutei appear to be specially sensitive and the Sartorius and Rectus femoris slightly so, this being the more remarkable as they are often more or less commingled, and the latter, as a part of the Quadriceps Extensor, a muscle almost always above the average in loss, would be expected to show a marked atrophy. The Hamstrings are always severely affected. Young animals do not appear to suffer more than old ones, unless they are very young and incompletely developed (No. 13). Further, the number of articulations affected seems to have more influence than any other factor in determining the severity of the atrophy.

The microscopic examination of the muscles is on the whole fairly in agreement with the notes of previous workers. Valtat[35] appears to have been misled by the occasional appearance of exceedingly atrophied fibres into considering the rest normal, but, as I have already remarked, all are changed, a few being larger but the vast bulk smaller, and this is a change easily recognisable without recourse to mensuration. The youngest animals, however, show far severer changes; both forms of striation are lost and the muscles appear to be on the road to complete atrophy, the sarcous matter having in places all but disappeared.

In the adult or semi-adult bones though changes in the direction of increased fragility were observed, no microscopic abnormalities could be detected. A very noteworthy point is, however, that without exception the diseased limbs were

decalcified quicker than the normal ones. The changes in the undeveloped bone are, however, suggestive and interesting.

Raymond having demonstrated that irritation of a peripheral nerve gave rise to the exaggerated reflexes and other phenomena somewhat similar to those produced by an arthritis, one rather looked for lesions in the posterior spinal ganglia such as have been described by Fleming.[105] None, however, could be found of the characteristic type.

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PART III.

Chapter I.

Pathological Physiology

There have been no lack of theories expressed as to the mode of action of the disease of the joint on the muscles. The principal are as follows;-

(1) Rest (2) Sympathy (3) Diminished blood supply due to swelling of joint (4) Absorption of nutrition of muscle by the joint (5) Progressive neuritis from nerve of articulation to nerve of muscle (6) Inflammation of the muscle (7) Reflex through the Vaso-motors (8) Toxaemia from the joint exudation (9) Reflex trophoneurosis.

(1) Rest: This is one of the oldest and one of the most persistent, first started by Hippocrates and still supported by some. I have already detailed the arguments of Brackett[71] the principal upholder of this theory. Would rest alone with no joint injury produce such an atrophy? I am not aware of any experiments made to settle this question but clinically one can say certainly not. Virchow has reported cases of 30 years immobility without atrophy, cases I believe of hemiplegia. Now hemiplegia lends itself open to some criticism in this matter the reflexes are exaggerated the nerve cells are incessantly receiving impulses from above through the irritated crossed Pyramidal tract and to some extent at least this must counteract the

immobility; the other limb may be referred to but here is no absolute immobility. On the other hand amyotrophy not unfrequently occurs in Hemiplegia in some cases early in others late, the exact pathological mechanism of which is not yet clear for some cases have been reported with central lesion (Brissand[87]) and others without (Babinski[88]) A much better example is forthcoming that of the child placed in long splints for Hip Joint disease; here both limbs are placed under identically the same conditions but the one buttock we find flat and wasted and the other normal in configuration. But another potent argument can be used, the rapidity of the atrophy is astounding, in four or five days a pronounced change is visible. Moreover in animals no fixation is employed and yet profound atrophy occurs while according to Brackett[71] the atrophy being proportionate to the degree of fixation none should have occurred.

(2) Sympathy;- This, the theory of John Hunter, may be considered perhaps as the forerunner of the modern reflex theories. Taken absolutely as it stands it is of course absurd but as a symbol of Hunter's insight and thoughtfulness it is indeed remarkable.

(3) Diminished blood supply due to swelling of the joint and direct pressure on the muscle can be dismissed very abruptly as it could only account for a very small proportion of cases.

(4) Absorption of nutrition by the joint:- One

would have to assume that a joint, such as the ankle, which when irritated is able to influence the nutrition of the entire limb, is able to absorb the nourishment intended to supply that limb, in man an amount approaching one fifth of the entire mass of blood. Far less than this would be quite beyond its power.

(5) Progressive neuritis from the nerve to the articulation to the nerve to the muscle. This has never yet been found in spite of the many clinical and experimental cases carefully examined for it.

(6) Inflammation of the muscle is of course found from time to time but the great majority show no such inflammation . Clinically no microscopic examination is necessary to prove its absence as a myositis of an entire limb would be provocative of intense pain swelling and other evidences of its presence.

(7) Reflex irritation of the Vaso-motors is proved not to be the cause because simulation of the Vaso-motor fibres leads to only very slight changes in the colour of the muscle and can only very slowly lead to atrophy. Further the neurosis associated with vaso-motor spasm show no amyotrophy.

(8) Local toxaemia from the joint exudation. This was a theory of Hoffa's [181] and led to some experiments I have not yet referred to. He injected cinnabar and other pigmentary agents into the knees of rabbits and then massaged but he only found the pigment in the

deeper layers of the muscles and as a rule only among the fasciculi of the subcrureus, never in the rectus itself. Hence he concludes that this is not the cause of the atrophy. As a matter of fact his experiments were unnecessary as with an inflammation of the knee the glutei are always affected and they are quite out of reach of a local toxaemia and a general one would affect of course the whole Muscular System. [101] Klippel

(9) A Reflex tropho-neurosis:- of strong probability previously this theory gained great ground from the experiments of Deroche [66], Raymond [67], and Hoffa [81] in dividing the posterior nerve roots. The experiments of Kornilow [77], of course, are against them but it is very probable from my own experience that some affection of the anterior roots has followed his sections. Haemorrhage after the operation comes on with reaction not infrequently and the animals die or get well in the latter case with clot in their medullary canal to upset their operators.

If this be a reflex condition all the phenomena noted clinically and experimentally can be explained. By no other single means can the hypertrophy or atrophy of the hair, the hypertrophy of the fat the signs of degeneration in the nerves and the muscular and Bony changes be satisfactorily accounted for. Such universally spread changes point to one thing and that is change in the central nervous system.

In at least two of my cases organic changes have

been found, but in five others perfectly comparable and equally carefully examined no change whatever has been found, so that it is clearly impossible to attribute the peripheral lesions to organic central change in every case, and it only remains to apprise the value of the changes found. The first (case 1) is a change in the axis cylinder, its nature I have already discussed fully and in spite of the opinions of very able men, who, it must be remembered have not had my opportunity of thoroughly searching the matter through and thinking it out, I believe it represents a physiological condition.

The second (case 6) in the whole lumbar region one abnormal cell is found, I think it may be dismissed.

The third represents an entirely different phase of affairs; we have a more or less circumscribed inflammatory area, extending over three or four vertebral segments. The change is one which would be quite easily recognized by ordinary processes with Haematoxylin or Carmine and one can therefore assume that this case is an exception to all those examined by Valtat[35], Deroche[66], Raymond[67], Duplay and Cazin[72] etc. There is no reason to suspect sepsis (unless from some abdominal organ and this is unlikely because the general appearance and behaviour of the animal was exceedingly good) . No neuritis is found. Therefore on this assumption one is forced to conclude that the irritation from the joint can in some very rare cases give origin to organic changes in the cord, and these can arise from aseptic

arthrites.

Ferrier[76] has suggested that in all cases there is a lesion of the dendrons of the cell. Unfortunately with specimens prepared with Nissl's method it is impossible to follow them for any distance as the sections must be thin or they will not stain evenly.

The Bichromate of Silver method as recommended by Roman Y. Cajal[97] is unfortunately too uncertain in its action when used on adult material to be used in a research where time was a matter of importance.

Further evidence of a possible organic change being produced is found from the case Gowers[73] reports where an arthritis of knee and ankle of one leg formed the probable source of origin of a Primary Spastic Paraplegia.

Chapter II.

General Considerations.

The experiments of Raymond[67] are of exceeding interest in other regions of neural physiology than those strictly limited to our subject. The result of division of the crossed pyramidal tracts (i.e. a hemisection) was to greatly accelerate the atrophy produced by an anthritis whereas the removal of the cortex and division of the posterior nerve roots alone sufficed to determine an atrophy . This in apparent contradiction to the fact that division of the posterior roots will prevent an atrpphy. This is explained by the fact that the tone of a nerve centre is maintained by the stimuli it receives from various others so long as, in this case, one is intact, the centre maintains its integrity, but the moment both are destroyed the tone falls and it loses its trophic capacity to a serious extent. It is to be noted that Raymond states the knee reflexes of his section of the cord do not disappear but more recent observers have shown they do disappear after a certain time to return in a few weeks and become exaggerated. Bastian has shown that in man complete division of the cord is followed by loss of reflexes but that the loss is immediate and is not recovered from and Sherrington

has explained this difference as follows; "The real difference I take it between tne Spinal Physiology of the Laboratory animals and that of man is that the spinal affluents do not of themselves suffice to keep metabolism of the spinal cells sufficiently braced up for the production of that muscular tone which is the substance of the jerk phenomenon; in other words the cerebral complement of spinal tonus is as compared with local or spinal complement disproportionally greater. In conformity with this it has been pointed out by Bastian that the superficial reflexes are usually in man extremely feeble after transverse lesion of the spinal cord, instead of being exaggerated as in the Laboratory experiments."

I have already alluded to the fact that some authors consider the atrophy is invariable that others think it is variable in certain particular conditions, and that others treat it entirely as a rarety. Some consider it as the result of a neurosis while others think the rheumatic is more subject to the affection.

Whatever views are taken, I think anyhow it will be granted that certain cases are brought before us which present to a more marked degree than others the phenomena in question. Why this? Why also does the Laboratory animal show a pronounced atrophy after an injury which to the naked eye leaves very little as traces? I think the remarks made at the commencement of this chapter will throw light on this question. The Laboratory animal suffers because the cerebral complement to his spinal

tone is small and the stimuli which he should receive from the periphery are exaggerated and perverted, hence the atrophy. Deroche[66] considered that the centre supplying the joint was the only centre injured and that its muscles were the only muscles affected; this is not so, we see the shock from the ankle is sufficient to affect the gluteal muscles very severely, and we may therefore probably conclude that the centres are each united closely besides the long afferent fibres which extend over and communicate with several segments. Now probably the individual who suffers largely from this atrophy belongs to a type which, though, by no means necessarily of a low mental type yet is of a type more closely allied to the animal type than the bulk of men are.

Marcus Beck[84] (Ericksen) quotes it as occurring in men who indulge in excessive sexual intercourse and in neurotic women. Now both of these may be considered to be on a low biological plane and their spinal cord to be in a low state of nutrition and very readily upset.

CHAPTER 111.

TREATMENT.

The discussion of the treatment can readily be reduced to small dimensions.

The more motion allowed the more risk of continuing the arthritis therefore the joint must be as perfectly as possible, immobilised.

Should the effusion show signs of not clearing up quickly, the joint must be tapped an operation too much feared by surgeons for if it is possible to inject irritants aseptically it is quite possible to withdraw some excess of aseptic fluid the articulation could be afterwards washed internally with sterilised water. The removal of distention is of the greatest importance. The use of counterirritants must be avoided they only irritate still more the irritated nervous centre.

The early commencement of passive motion and massage will dissipate any inflammatory exudation round the joint.

The diathesis if marked requires careful treatment, the nervous and irritable who will lie and worry and fret must have sedatives, the gouty and rheumatic suitable diet and drugs.

A most important feature is the prevention of fresh strains when cure has fairly set in and while passive motion must be early commenced, voluntary motion and more especially any real work such as walking must be delayed. If atrophy has once commenced to a distinct degree, probably static electricity is the best, the muscles reacting much better to the electric spark than to any other form. If this is not available the constant current very slowly interrupted is recommended. Very weak currents left on several hours may be used care being taken to prevent ulcers forming beneath the electrodes. Afterwards should supports be found necessary properly articulated apparatus well laced up and not elastic bandages should be used.

BIBLIOGRAPHY.

In Chronological Order.

1	Hippocrates.	Littré French Translation. Vol. IV. pp. 79, 141, 231. " V. " 135, $79.	
2	Galen.		
3	Petit.		
4	Hunter.	Lectures on the Principles of Surgery. p. 518.	1835.
5	Nelaton.	Coxalgie: Bulletins de la Société Anatomique de Paris.	1835
6	Godin.	Bulletins " " "	
7	Coulston.	On the disease of the Hip Joint p.51	1837.
8	Tyrrell.	St. Thomas's Hospital Reports. Vol. 1.	
9	Bonnet.	Traité des Maladies des Articulations. t.i.	
11	Roux.	Annals de Chirurgie.	1845
10	Malgaigne J.F.	Traité des Fractures et des Luxations. T. ii.pp.19, 49.	1847
14	Charcot J.M.	" " "	
12	Cruveilhier.	Traité d'Anatomie Pathologique. T. III. pp.127, 128.	
13	Adams.	Treatise on Rheumatic Gout etc.	1857
15	Plaisance.	Thèse de Paris.	1858.
17	Bauer.	On Hip Disease. New York.	1859
16	Gubler.	Archives générales de Med.	1860-61
	"	Société de Biologie.	1861
18	Brown Sequard.	Philadelphia	1860

19	Vulpian.	Leçons sur la Physiologie Nerveuse	1866
21	Liègeois.	Traité de Physiologie	1869
20	Vergely	Thèse de Paris	1866
22	Ollivier	Thèse d'Aggrégation p.97	1869
23	Paget J.	Lectures on Surgical Pathology	1870
24	Le Fort	Société de Chirurgie	1872
25	Collette	Thése de Paris	1872
26	Beni-Barde	Société d'Hydrologie	1872
27	Sabomin	Thèse de Paris.De l'atrophie musculaire rhumatismale	1873
28	Weir-Mitchell	Des Lesions des Nerfs (French Translation)	1874
29	Vulpian (Weir-Mitchell)	" " "	1874
29a	Vulpian	Leçons sur les Vaso Moteurs T. ii. p.327	1875
30	Desnot et Barié	Note sur un cas d'atropie général du membre inférieur droit consecutive d'un traumatism Prog. Méd.p.557	1875
31	Le Fort	Société de Chirurgie Des paralysies avec atrophie aiguë de quelques groupes de muscles dans certaines affections articulaires et en particulier dans l'hydrarthrose	1876
32	Onimus	Société de Médecine	1876
33	Paget Sir J.	Clinical Lectures and Essays 2nd.Ed. p.216	1877 1879
34	Vulpian	Leçons de Pathologie rxpérimentale	1877
35	Vallat	Thèse de Paris Sur les atrophies musculaires en rapport des Arthrites.	1877

77	Kornilow	Société de Neurologie et de Psychiatrie de Moscow. Revue Neurologique	1893
78	Mansell Moullin	Sprains. p.59	1894
79	Huguet	Amyotrophies traumatiques Gazette des Hopitaux p.759	1894
80	Thevenet	Atrophies de plusieurs muscles de l'épaule consecutives d'une Pleurésie. Lyons Médical LXXV.	1894
81	Hoffa	Volkmann's Klinische Vortrage Chirurg No 13	1894
82	Plicque	Les Amyotrophies d'origine articulaire Gazette des Hopitaux p. 187	1894
98	Nissl	Mittheilung zur Anatomie des Nerven Zelle Allgemeine Zeitschrift für Psychiatrie No.50 p. 370	1894
91	Leloir	Contribution à l'étude des Atrophies musculaires d'origine spinale produites par les Lésions periphériques Progrès Médical p.804	1881
83	Moore,W.L.	Muscular Atrophy consequent on certain articular Lesions Occ. Med. Times Sacramento	1894
84	Ellenberger	Anatomie des Hundes	189-
	Erichsen	System of Surgery 10th Ed. Chap. 48.	1895
	Hyde,S.	Causes and Treatment of Rheumatic Arthritis pp.16,25	1896
96	Marinesco,G.	Des Polynevrites en rapport avec les lésions secondaires et les lésions primitives des cellules nerveuses Revue Neurologique	1896
	"	Les Polynevrites en rapport avec la Théones des neurones. Comptes Rendues de la Société de Biologie	1895

52	Vulpian	Deux d'atrophie musculaire Gaz. des Hopitaux. p.130	1883
53	Barker	Diseases of Joints, Holmes & Hulkes System of Surgery	1883
54	Trelat	Société de Chirurgie	1885
55	Duplay et Clado	Note pour servir à l'étude des altérations musculaires consecutives aux fractures. Progrès Médical. T.ii.p.69.	1885
56	Moussons	Thèse de Bordeaux	1885
88	Babinski	Atrophie musculaire d'origine cérébrale avec integrité des cornes antérieures et les nerfs motems. Société de Biologie.20.Fev.	1886
57	Parisot	Thèse d'Aggrégation . La Pathogênie des Atrophies musculaires.	1886
58	Desplats	Pleuresy and Muscular Atrophy. Semaine médicale. April 18.	1887
99	Flesch	Mittheilung der naturforscher Gesellschaft in Berne.S.192	1882
59	Metge	Thése de Bordeaux	1887
60	Barbillon	Thèse de Paris	1887
61	Macnamara C.N.	Diseases of Bones and Joints 3rd.Ed. p.328	1887
62	Strümpell	Ueber die nach Gelenkerkrankungen eintretende Muskel-Atrophie. Münchener Medicin. Wochenschrift.pp.206,211.	1888
63	Charcot,J.M.	Leçons des Mardi. p.336	1888
101	Klippel	Thèse de Paris Les Amyotrophies dans les maladies chroniques générales et leurs rapports avec lésions des nerfs périphériques	1888
86	Bezy	De l'atrophie musculaire comme cause des douleurs articulaires Prog.Méd. p.213	1889

51	Hilton	On Rest and Pain	1877
36	Darde	Thèse de Paris	1877
37	Berquiem	Thèse de Paris	1877
38	Urgy	" " "	1878
38[a]	Boequet	" " "	1878
39	Desplats	De l'atrophie musculaire dans la peri-arthrite scapulo humerale. Gaz. Hebdomadaire p.374	1878
40	Debove	Note sur les Atrophies musculaires d'origine articulaire. Prog. Méd. p. 1011.	1880
41	Picqué	Des Atrophies. Gaz. Med. de Paris. p. 255	1880
42	Christin	Thèse de Paris	1880
43	Batigne	" " "	1880
44	Descosse	" " "	1880
45	Vignes	" " "	1880
87	Brissand	" " "	1880
	Latasie	" " "	1880
46	Dubreuil	Leçons de Clinique chirurgicales.	1880
47	Guyon et Féré	Note sur l'Atrophie musculaire consecutive à quelques traumatismes de la hanche	1881
48	Guichard	Thèse de Bordeaux	1881
91	Leloir	Contribution à l'étude des atrophies musculaires d'origine spinale produites par les lésions peripheriques. Progrès Médical p.804	1881
49	Combescure	Thèse de Montpellier. Les atrophies musculaires d'origine periphérique.	1882
50	Charcot J.M.	Sur l'atrophie musculaire qui succède à certaines lésions articulaires. Prog.Med.p. 377	1882

64	Spender	Osteo-Arthritis. p.23	1889
65	Ord	" " (Spender) p.23	1889
89	Raymond	Les Atrophies Musculaires	1889
66	Deroche	Thése de Paris	1889
68	Allingham H	Internal derangements of the knee joint	1889
69	Ballet	La Coxalgie hystérique avec l'Atrophie musculaire Loc. Méd. des Hopitaux July 2.	1889
67	Raymond	Recherches expérimentales sur les amyotrophies consecutives aux Arthrites traumatique Revue de Médicine	1890
77a	Kornilow	Annalen der Gesellschaft für Chirurgie. Moskau No.3	1890
70	Dackschewitsch	Ein Fall von Muskel Atrophie bei Gelenkerkrankung Neurolog. Centralbl.June ii p. 353	
1oo	Friedman	Ueber degenerative Veränderungen der Ganglien Zellen bei akuter Myelitis Neurolog. Centralbltt.p.1	1891
71	Brackett	Atrophy in Joint Disease Boston Medical and Surgical Journal. No.24. Dec.	1891
72	Duplay et Cazin	Archives générales de Médecine	1891
73	Gowers.W.R.	Diseases of the Nervous System 2nd.Ed. Vol.II. p. 498	1892
74	Lane,Hugh	Differentiation in Rheumatic Diseases A A 9 p.39	1892
	Paterson	Journal of Anatomy and Physiology	1893&1894
75	Charcot,J.M.	Amyotrophies spéciales réflexes d'origine articulaires Progrès Médical p. 225	1893
76	Ferrier	Brit. Med. Journal	1893

	Marinesco, G.	Des lésions primitives et des lésions secondaires de la cellule nerveuse Société de Biologie	1896
95	Sadovsky, S.	Modification de la méthode de Nissl. Névrite experimentale par compression et lésions consecutive des centres nerveux Compte Rendue de la Société de Biologie	1896
105	Fleming	Some Notes on Ascending Degeneration (so called) and on the changes in nerve cells consequent there-on. Brit. Med. Journal Nos. 1, 2&3	1897
		Ed.med.Journal Nos. 1,2 & 3	1897
	Kraepelin	Psychiatrie 5te.Aufl. S.103	1896
85	Bannatyne	Rheumatoid Arthritis p.29	1896
102	Sherrington	The Physiology of the Spinal Cord St. Thomas's Hospital Reports	1894
92	Gunther Carl	Backteriologie p.395	1895
103	Sherrington	Brain January	1896
94	Lenhorsch	Der feinere Bau des Nerven-Systems	1896
97	Ramon J Cayak	Nouvelles idées sur le système nerveux	1896
104	"	Archiven für Psychiatrie und Nervenheilkunde.	1897